FM 8-283
NAVMED P-XXXX
AFJMAN XX-XXX
MCRP X-XXX

TREATMENT OF NUCLEAR WARFARE CASUALTIES AND LOW-LEVEL RADIATION INJURIES

INITIAL DRAFT

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IT DOES NOT CONTAIN ARMY-APPROVED DOCTRINE.

HEADQUARTERS, DEPARTMENT OF THE ARMY

US ARMY MEDICAL DEPARTMENT CENTER AND SCHOOL
FORT SAM HOUSTON, TEXAS 78234-6175

APRIL 2000

S: 30 May 2000

MCCS-FCD-L (25-30xx)

MEMORANDUM FOR SEE DISTRIBUTION

SUBJECT: Staffing of Field Manual 8-283, Treatment of Nuclear Warfare Casualties and Low-Level Radiation Injuries (Initial Draft)

1. Reference Army Regulation 25-30, The Army Integrated Publishing and Print Program.

2. Provided for your review and comments is the initial draft for FM 8-283, Treatment of Nuclear Warfare Casualties and Low-Level Radiation Injuries. It is available for download at http://dcdd.amedd.army.mil/index1.htm. This manual will be a multi-service publication when completed. The publication numbers for the other services will be provided during the staffing process.

3. This manual is part of the ongoing Army effort which parallels the NATO NBC Medical Working Group initiative that separates FM 8-9 into three FMs: 8-283, 8-284, 8-285 (N, B, & C, respectively). This draft has been prepared with assistance by personnel from the Armed Forces Radiobiology Research Institute and the Academy of Health Sciences, AMEDD Center and School.

4. Request your input in subject areas you feel need to be included. Request your comments be directed to material that is technical in nature. Editorial review will be conducted by the action agency.

5. To enable us to meet the established production schedule, request your comments and recommendations not later than 30 May 2000.

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6. Point of contact for this action is Mr. Doug Hanson, DSN 471-9340; Commercial (210) 221-9340; FAX 471-9662. E-mail: Douglas.Hanson@cen.amedd.army.mil.

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DEPARTMENTS OF THE ARMY, THE
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COMMANDANT MARINE CORPS
Washington, DC DATE)

TREATMENT OF NUCLEAR WARFARE CASUALTIES AND LOW-LEVEL RADIATION INJURIES

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PREFACE

Purpose

This publication serves as a guide and a reference for trained members of the Armed Forces Medical Services and other medically qualified personnel on the recognition and treatment of nuclear warfare casualties and low-level radiation injuries.

Scope

a. This publication--

(1) Classifies and describes potential nuclear and low-level radiation threats and hazards.

(2) Describes the scientific aspects of ionizing radiation and its effects on organs and systems of the body.

(3) Describes the effects of blast, thermal radiation, and nuclear radiation on organs and systems of the body.

(4) Describes procedures for first aid, medical diagnosing, treating, and management of nuclear warfare and low-level radiation casualties.

b. The material in this publication is applicable to both the nuclear battlefield and to operations where a low-level radiation hazard exists; this includes military support to United States (US) civilian agencies during weapons of mass destruction (WMD) consequence management operations.

c. The treatment modalities contained in this manual are based upon those described in the most recent North Atlantic Treaty Organization (NATO) Handbook on the Medical Aspects of NBC Defensive Operations AMedP-6(C), Study Draft IV, dated 29 September 1999.

d. The use of the term “level of care” in this publication is synonymous with “echelon of care” and “role of care.” The term “echelon of care” is the old North Atlantic Treaty Organization (NATO) term. The term “role of care” is the new NATO and American, British, Canadian, and Australian (ABCA) term.

Standardization Agreements
This manual is in consonance with NATO Standardization Agreements (STANAGs) 2475, Planning Guide for the Estimation of NBC Battle Casualties (Nuclear), AMedP-8 (A), Volume. I.

User Comments
Users of this publication are encouraged to submit recommendations to improve the publication. Comments should be keyed to the page, paragraph, and line(s) of the text where the change is recommended. The proponent for this publication is the US Army Medical Department Center and School (AMEDDC&S). Comments should be forwarded to: **Commander, AMEDDC&S, ATTN: MCCS-FCD, 1400 East Grayson Street, Fort Sam Houston, Texas 78234-6175**.

Gender Statement
Unless this publication states otherwise, masculine nouns and pronouns do not refer exclusively to men.

Use of Trade Names/Trademarks
Use of trade names/trademarks in this publication is for illustrative purposes only. Their use does not constitute endorsement by the Department of Defense (DOD).

References
References listed should be consulted for details beyond the scope of this publication.

CHAPTER 1

INTRODUCTION

1-1. The Threat of Nuclear Warfare and Low-Level Radiation Against United States Forces and Civilian Populations

In the post-World War II environment, there were two basic scenarios for an exchange of nuclear weapons: Either the limited use of tactical nuclear weapons on a European or regional battlefield, or a general strategic exchange of large-yield thermonuclear weapons. The medical response for casualties in the limited tactical nuclear weapons scenario would probably have consumed all available medical resources. Nuclear radiation casualties would have lived or died, depending on the dose received. Radiation victims of a general strategic exchange of large-yield thermonuclear weapons would have been regulated to the end of the line. Casualties would have been treated with whatever was left intact after the massive destruction of cities and the medical infrastructure. Today, the most likely threat is nuclear accidents involving medical or industrial radiological material, followed by terrorism involving the release of a radiological dispersal device (RDD), a single nuclear detonation, and then tactical and strategic nuclear war scenarios (see Figure 1-1).

Figure 1-1. Terrorist actions and accidents are more likely today.

a. Nuclear Weapons. Nuclear weapons are normally thought in terms of an explosion and the resulting mushroom cloud. This is associated with the type of weapon that was used at Hiroshima and Nagasaki, resulting in blast effects, heat, nuclear radiation, and fallout. Such an explosion occurs when enough fissile material, either uranium or plutonium, is compressed into a given volume to cause supercriticality. This is a state in which each neutron that is produced in the fuel generates a net of more than one neutron via the fission process.

(1) *Fission weapon.* Because of the stray neutrons produced in the environment by spontaneous fission and neutrons present in the atmosphere from cosmic ray interactions, as well as others generated in various ways, a critical or supercritical mass may be unintentionally created. This could result in the nuclear material melting or possibly exploding. It is necessary, therefore, that, before detonation, a nuclear weapon contain no piece of

fissionable material as large as a critical mass. At the time of the detonation, some method must be employed to make the mass supercritical by changing its configuration. Two general methods have been developed for quickly converting a subcritical mass into a supercritical one:

- *The gun-type weapon*. This type of weapon is essentially a tubular device in which a high explosive is used to blow one subcritical piece of fissionable material from one end of the tube into another subcritical piece held at the opposite end of the tube.

- *The implosion-type weapon*. This method uses a subcritical mass of uranium-235 (^{235}U) or plutonium-239 (^{239}P) that is compressed to produce a mass capable of supporting a supercritical chain reaction. This compression is achieved by the detonation of specially designed high explosives surrounding a subcritical sphere of fissionable material.

(2) *Enhanced radiation weapon.* An enhanced radiation (ER) weapon, by special design techniques, has an output in which neutrons and X-rays are made to constitute a substantial portion of the total energy released. For example, a standard fission weapon's total energy output would be partitioned as follows: 50 percent as blast; 35 percent as thermal energy; and 15 percent as nuclear radiation. An ER weapon's total energy would be partitioned as follows: 30 percent as blast; 20 percent as thermal; and 50 percent as nuclear radiation. Thus, a 3-kiloton (KT) ER weapon will produce the nuclear radiation of a 10-KT fission weapon and the blast and thermal radiation of a 1-KT fission device (see Figure 1-2). However, the energy distribution percentages of nuclear weapons are ultimately a function of yield.

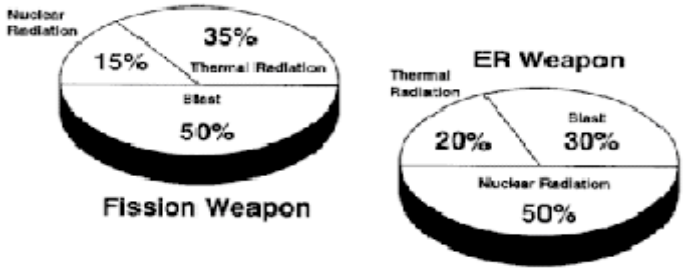

Figure 1-2. Weapon energy distribution.

(3) *Fusion weapon*. In general, fusion may be regarded as the opposite of fission. It is the combining of two light nuclei to form a heavier nucleus (thermonuclear reaction). Fusion reactions are used to increase the yield by making more efficient use of the nuclear material. The only practical way to obtain the temperatures and pressures required is by means of a fission explosion. Consequently, weapons with fusion components must contain a basic fission component. One way to boost the fission yield is by incorporating thermonuclear reactions into the design of the weapon. Introduction of neutrons from thermonuclear reactions at the time of supercriticality of the fissile material can provide the required boost. The usual fusion material used for this purpose is a mixture of deuterium and tritium gas. In order to

greatly increase the yield of nuclear weapons, thermonuclear and/or second stages are designed and developed. To do this, designers have to obtain and master hydrodynamic computer programs that correctly describe regimes of extremely high temperatures and pressures. The materials used for the weapon's second stage are lithium (and/or lithium deuteride), tritium, and deuterium.

b. Isotopic Weapons. Many communities have factories and hospitals with significant amounts of radioactive material. Radioactive materials common in a factory or hospital could be scattered as the result of a conventional explosion, thereby exposing a large number of people. This type of isotopic weapon is known as a RDD. Anyone trapped inside a target structure and their rescuers would be internally and externally contaminated, although there will be no initial high-energy radiation and no fallout of fission products. An explosion near a nuclear medicine facility's cobalt-60 source, for instance, would spread the radioactive material throughout the rubble of the target structure. There would also be conventional blast and burn injuries as well as exposure to radiation from the contaminants. Therefore, every firefighter, rescuer, and casualty in the immediate area could be exposed to radiation.

c. Nuclear Accidents and Incidents.

(1) *Chernobyl*. In Russia, the nuclear reactor explosion in Chernobyl in 1986 was a disaster of immense proportions as was the contamination of the Technya River basin. In addition, the Russian navy has a host of deteriorating submarine reactors.

(2) *Three Mile Island* (Figure 1-3). In the United States (US), we are more at risk from the vast amount of medical and commercial radioactive material than from events like the Three Mile Island incident on March 28, 1979. Although the event was covered extensively by the media, the amount of radionuclides released was actually very small and the safety measures in place to halt the meltdown worked.

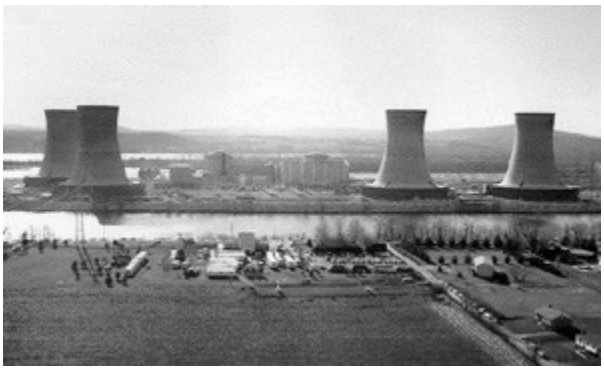

Figure 1-3. Three Mile Island facility.

(3) *Other Incidents*. Rather than major accidents, the greater risk is actually from incidents like that in March 1996 when 21 Texans were exposed to up to 0.3 Gy of radiation from an abandoned, and then stolen cobalt-60 industrial unit. Examples of other incidents include:

- In Brazil, a medical radio-cesium unit was dismantled, exposing 21 people, three of whom died.

- Radiation detectors went off when a truck loaded with steel entered a monitored nuclear facility. A junk dealer had sold an old cobalt-60 radiotherapy machine as scrap metal, which was made into good quality, but radioactive, cobalt steel.

1-2. Physical Principles of Ionizing Radiation

a. Nuclear energy is generated from a change in binding energy within the nucleus. A thermonuclear fission-fusion device releases more energy than a pure fission device. Only 15 percent of energy released in a nuclear explosion is in the form of radiation; 85 percent is as heat and blast. While heat and blast are the same forms of energy regardless of the source, there is exponentially more of each in a nuclear explosion (see Figure 1-4).

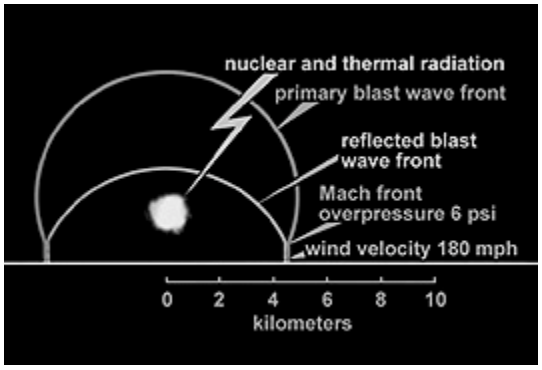

Figure 1-4. Energies released in a nuclear explosion.

b. Radiation is in several forms--charged particles, neutrons, and gamma rays. Of the three, charged particles (that is, alpha and beta) are the only form stopped by as little as normal clothing unless the source is internalized. In that case, the radiation threat depends on the type of source. The amount of absorbed radiation is measured in Gray (Gy). Its biologic effectiveness is the Sievert (Sv). Previously used equivalents were the radiation-absorbed dose (rad) and the roentgen-equivalent in man/mammal (rem) (1 Gy = 100 rad; 1 Sv = 100 rem). See Chapter 3 for a detailed discussion of units of measure.

1-3 Immediate Causes of Death

Most persons close enough to a nuclear explosion to develop radiation injury symptoms within 1 to 2 hours of the detonation would be killed by the blast wave, the thermal pulse, or the resulting fires. Most who are picked up by the blast winds will suffer significant impact injuries.

a. *Blast Injuries*. Blast injuries are caused by the overpressure wave that travels outward from ground zero at several hundred kilometers (km) per hour. A 1-megaton (MT) weapon creates a static overpressure of 6 pounds per square inch (psi) and dynamic overpressures, or winds, of 300 km per hour at 4 km from ground zero. Biologic systems are more resistant than physical structures to overpressure, but they are also subject to injuries from flying objects, being impaled upon stationary objects, or blunt force trauma from the body impacting on immobile objects.

b. *Thermal Injuries*. Nuclear weapons emit electromagnetic waves across the spectrum. Infrared waves, traveling at 300,000 km per second (the speed of light), will set fire to virtually any combustible material. A person who looks directly at the flash will suffer burns of the retina. Such burns may occur 10 miles from the detonation depending upon the yield. Someone who looks in another direction will be flash blinded, which is a temporary condition. Thermal injuries are burns regardless of their etiology. Because dark colors absorb heat and then ignite, and because light colors reflect heat, the burns will exhibit the patterns of the dark clothing worn at the time of detonation.

1-4. Radiation Injuries

Radiation injuries are determined by the type and amount of radiation exposure, and shielding, if any. Most radiation medicine data are based on controlled dosages to specific parts of the body, or data are based on carefully modulated whole-body dosages. Exposure after the detonation of a nuclear device, or an exposure from RDD is neither controlled nor modulated.

a. Acute radiation syndrome (ARS) has three subsyndromes which are progressive and dose dependent. They are the hematapoietic, gastrointestinal (GI), and cardiovascular (CV)/central nervous system (CNS) syndromes. Generally, the radiation dose received determines the onset of nausea and vomiting; that is, the sooner the onset of symptoms and the longer they last, the higher the dose received.

b. Acute radiation syndrome patients go through four stages: Prodrome, latent, manifest illness, and recovery or death. While many factors determine how each patient fares, the better the patient's health to begin with, the better the chance of recovery. Table 1-1 shows the approximate dose for the ARS for total-body irradiated, untreated victims. Treatment should increase survival up to about 10 Gy. Partial shielding and aggressive therapy may result in survivors estimated to have received 20 Gy. Those exposed to 30+ Gy, even with supportive care, will probably not survive. The prime patients in intensive care beds will be those with lower exposures.

Table 1-1. Acute Radiation Syndrome

Syndrome/Dose	Prognosis
Hematopoietic	
0-2 Gy	Excellent
2-6 Gy	20%-30% death
Gastrointestinal	
6-10 Gy	80%-100% death
10+ Gy	100% death
CV/CNS	
30+ Gy	Certain death

c. The following are ARS symptoms:

(1) Hematopoietic syndrome will show an initial lymphocyte drop, which is followed by agranulocytosis. The higher the dose, the steeper the drop. Absolute lymphocyte counts, during the first 3 days after exposure, are the best predictor of patient dose.

(2) In GI syndrome, the gut mucosa is injured, then sloughs and shuts down. This exposes the patient to translocation of bacteria. Since this happens at the same time that the granulocyte count falls, sepsis and opportunistic infections complicate the effects of fluid and electrolyte losses.

(3) Cardiovascular and CNS changes are probably due to microvascular leaks. These patients will almost certainly die.

1-5. First Aid

First aid for nuclear casualties will focus on treating conventional blast and thermal injuries. This consists of performing the standard lifesaving measures and then treating the conventional injuries such as head wounds, fractures, and burns. There are no direct first aid measures for suspected radiological casualties. Therefore, personnel exposed to low-level radiation without any conventional injuries should be transported to the nearest military treatment facility after decontamination as appropriate. Casualties of nuclear incidents may have combined injuries, and these personnel should receive standard first aid for conventional injuries as required.

1-6. Triage

a. Radiation exposure levels can be estimated based on the onset and duration of certain symptoms (see Figure 1-5). In the past, when a patient was exposed to an estimated dose of 6 Gy, treatment was expectant, but this no longer applies. With aggressive therapy, it is

possible to anticipate survival at 10 Gy and a few survive after 20 to 30 Gy of partial-body exposure.

Exposure Potential

Symptoms	Unlikely	Probable	Severe
Nausea	-	++	+++
Vomiting	-	+	+++
Diarrhea	-	+/-	+/- to +++
Hyperthermia	-	+/-	+ to +++
Erythema	-	-	- to ++
Hypotension	-	-	+ to ++
CNS dysfunction	-	-	- to ++

- = Absent
+ = Present
++ = Excessive
+++ = Very Excessive

Figure 1-5. Exposure potential.

b. A triage estimation of radiation injury in these casualties can be made based upon history and symptoms (that is, the severity of nausea and vomiting). The presence of neurological signs usually indicates a nonsurvivable injury. Serial lymphocyte counts in the first 48 hours helps confirm irradiation insult; the initial response to exposure is a drop in the count. A drop of more than 50 percent in the absolute lymphocyte count indicates a significant radiological injury. Therefore, the steeper and more profound the drop, the higher the radiation dose received. While immunocompromise indicated by the drop is the initial killer in exposure incidents, it is also the effect most amenable to aggressive treatment.

1-7. Contamination and Exposure

a. External Contamination. External contamination will occur when radioactive particles accumulate on the surface of skin and clothes. Although such particles are usually alpha-beta emitters, they can cause substantial local injury if not washed off. Simple decontamination procedures, such as removing patient's clothing and then washing the patient with soap and water, are 95 percent effective.

b. Internal Contamination. If the radioactive material is an alpha emitter, such as plutonium, then the prime danger will be inhalation and ingestion of the dust. The casualties and rescuers in the immediate area will almost certainly inhale a significant quantity of the nuclide long before they know they are exposed. Internal contamination means continuous exposure to the organs until the radionuclide is eliminated from the body or undergoes complete decay (see paragraph 1-8).

c. *Exposure to Radiation.* Irradiation occurs when the subject is in the presence of an active source that emits a sufficient quantity of neutrons or gamma rays from, for instance, a cobalt therapy device, or an extremely high level of ground contamination. Individuals exposed to significant radiation doses over a protracted period of time would not be radioactive but would suffer from ARS. Hospitals and clinics would note a substantial number of patients with acute gastroenteritis and no lymphocytes. Individuals exposed to only 0.5 to 1 Gy might be relatively asymptomatic; but over a period of a few weeks, a large percentage would develop endemic diseases.

1-8. Internal Contamination

a. A patient whose body contains a radionuclide is considered internally contaminated. That patient will continue to receive additional exposure until the radionuclide either completely decays or is removed. The two primary sources of internal contamination are accidents and medical misadministrations. A third source is introduction of radionuclides through wounds. All of the transuranic elements are heavy metals and the base metals or oxides of these metals are insoluble and must undergo hydrolysis to be absorbed and circulated. Salts, such as uranyl fluoride, are immediately systemic, excretable, and toxic. The extent of internal radiation contamination is determined by the following factors:

- Amount of radionuclide(s).
- Energy and type of radiation.
- Length of time in the body.
- The critical organ affected.

b. The three stages of internal contamination are intake, metabolization, and elimination. The route of entry and the state of the element determines toxicity. For example, the decay rate of inhaled/exhaled tritium gas is insignificant. Yet tritium ingested as tritiated water is absorbed through the lungs, the GI tract, and the skin, and immediately equilibrates with total body water. When a radionuclide enters the human body, the body reacts exactly as it would to a nonradioactive isotope. As far as the body is concerned, tritium and hydrogen are identical as are iodine-127 and iodine-131. The target organ of the specific nuclide depends primarily on the organ's metabolism. Treatment is directed toward preventing deposition of a hazardous quantity and moving the nuclide out of the body by speeding up the metabolic processes through cathartics, dilution, and chelation.

c. Medical care providers need to assess the patient's level of contamination and the location of contamination. Procedures and resources to evaluate the type and quantity of internal contamination are described in Section VII of Chapter 6. These assessment procedures to determine exactly how much contamination has been internalized is often a time-consuming process (see Table 1-2).

Table 1-2, Guidelines for Bioassay Sampling

Optimum Sampling Time After Exposure

Suspected Radioactive Material	Optimum Sample Time		Sample Quantity
	Feces	Urine	
Plutonium	24 hours	14-21 days	24-hour total
Uranium	24 hours	24 hours	24-hour total
Tritium	N/A	12 hours	1 voiding

d. Half-life is defined as the time required for half of the atoms of a given sample of radioisotope to decay. Half-life values range from fractions of a millionth of a second to billions of years. The biologic half-life of an internalized radionuclide is determined by normal chemical and metabolic processes such as urination, respiration, desquamation, GI motility, and phagocytosis. The physical half-life is determined by the isotope and its intrinsic rate of decay.

1-9. Combined Injury

Radiation exposure and physical trauma combined are worse than either injury alone. The change in lethality is dramatic when even nonlethal doses of radiation combine with severe burns. A geometric progression will be typical in victims of terrorist attacks in which many casualties suffer combined injuries.

a. The progressive effect of conventional wounds and irradiation can be reversed if all wounds are closed early. In experiments, the procedure resulted in no increase in lethality and even improved recovery when compared with recovery from radiation injury alone. This effect is probably due to the metabolic response to trauma and the elimination of the nidus of infection prior to leukopenia. Therefore, primary closure during surgical intervention involving radiation-combined injury must be completed in the first 48 hours after irradiation. This is a marked change compared with the timing of surgery for routine trauma. Reconstructive surgery must be postponed for two months until complete resolution of the radiation injury. Surgery during the exclusion period will result in excessive mortality.

b. Because radiation injury is not immediately life threatening, initial care of a combined injury casualty should address conventional injuries like burns and wounds. After emergency procedures for ventilation, perfusion, and hemorrhage, the casualty should be stabilized. Radioisotope decontamination should precede emergency surgery, which should be followed by definitive care and treatment of radiation injuries.

1-10. Treatment of the Radiation Casualty

Initial therapy is directed toward fluid and electrolyte balance, maintenance of nutrition, and alleviation of the granulocytopenia. If blood component therapy is required, all blood products must be irradiated with 20 Gy prior to infusion to prevent graft versus host disease.

a. Infection. At 3 weeks, untreated opportunistic infections become paramount. Infection may occur for the following reasons:

- Oropharyngeal respiratory tree colonization.
- Wound contamination.
- Intestine colonization.
- Artificial invasive devices.
- Profound immunosuppression.
- Pathogens in environment.

Infection should be managed with reverse isolation precautions and specific broad-spectrum anti-microbial therapy as per immune compromised patient protocol. All invasive monitoring is precluded. A long-term central line is a death sentence. Therapy must be directed toward infection prevention and shortening the period of relative immuno-incompetence. Every deep vein catheter is a probable disaster. Direct enteric feeding is safer than total parenteral nutrition (TPN) and stimulates regeneration of the intestinal mucosa; it should be started as early as possible.

b. Partial-Body Exposure. Partial-body exposure is more likely in instances of battlefield exposure, accidental radiation release, or terrorist action. Stimulation of the remaining stem cells to recolonize the marrow dramatically increases the reproduction of blood elements. However, such patients are not good candidates for bone marrow transplant because the surviving marrow induces host versus graft disease (see Figure 1-6).

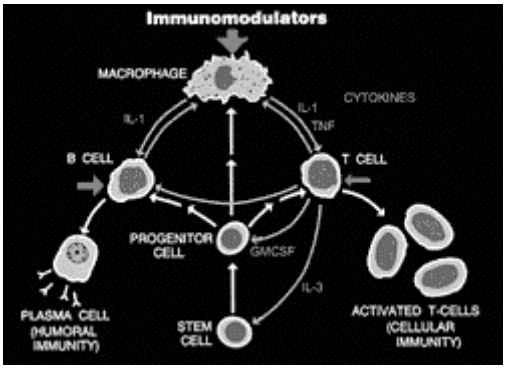

Figure 1-6. Immunomodulators.

c. *Cytokine Therapy*. The most important new therapy is the use of cytokines such as the two approved for use in bone marrow transplant patients. These two are the granulocyte macrophage-colony stimulating factor (GM-CSF) and the granulocyte-colony stimulating factor (G-CSF). Others under evaluation include megakaryocyte growth and development factor/thrombopoietin (MGDF/Tpo) and the Interleukin 3 (IL-3) agonist, Synthokine. When combined with the probability of partial shielding, cytokine therapy should change accidental irradiations at dosages once considered 100 percent deadly to a nonlethal injury. The current recommendation is to initiate cytokine therapy as soon as possible so as to decrease the period of absolute neutropenia, thereby reducing the risk of infection during recovery from marrow aplasia.

CHAPTER 2

THE NUCLEAR THREAT

Section I. NUCLEAR AND RADIOLOGICAL WEAPON TECHNOLOGIES

2-1. General

The proliferation of nuclear material and technology has made the acquisition and adversarial use of nuclear weapons and RDDs more probable. Military personnel and the civilian population will expect that state-of-the-art medical support will be employed to treat casualties from the use of these weapons. Treatment protocols for radiation casualties are now effective, practical and possible, and must be part of US Armed Forces medical contingency planning efforts. The following paragraphs provide an overview of the nuclear threat facing US military personnel and civilian populations. These threats include accidental exposures to radioactive material, RDDs, destruction of reactors, limited use of tactical nuclear weapons, and the least likely occurrence--general thermonuclear war.

2-2. Nuclear Weapons

To produce a nuclear explosion, a weapon must contain an amount of uranium or plutonium that exceeds the mass necessary to support a critical chain reaction, that is, a supercritical mass (see Chapter 3). The nuclear material must also be in the right configuration so that successive generations of neutrons can cause equal or increased numbers of fissions. To obtain a significant nuclear yield of the nuclear explosive, sufficient neutrons must be present within the supercritical core at the right time. If the chain reaction starts too soon, the result will be only a "fizzle yield," much below the design specification; if it occurs too late, there may be no yield at all. Several methods can be used to make a mass of fissionable material supercritical.

- The active material can be purified to eliminate unwanted chemical impurities that might otherwise absorb neutrons.

- Fissionable material can be enriched, that is, the amount of ^{235}U as compared to ^{238}U can be increased.

- The material can be machined into the most efficient shape. A spherical shape can be employed to provide the greatest volume with the least surface area, thereby reducing the probability of neutron loss.

- Moderators can be used to slow down fission neutrons, increasing the probability of the material producing fissions.

- Finally, neutrons that have escaped the active material can be reflected back by using suitable materials as reflectors. Reflectors, used as tampers, can also physically delay the

expansion of the exploding material allowing more fission to occur, thereby resulting in an increase in explosive energy.

2-3. Isotopic Weapons

a. Another type of nuclear weapon--the isotopic weapon--is a contamination or a RDD. Isotopic weapons are inexpensive and require no advanced technology to fabricate. They are ideal weapons for terrorism and are used to intimidate and deny access to an area by spreading radioactive material. The use of isotopic weapons is a more likely event than other scenarios, such as strategic/theater nuclear war. The only more likely event than the use of an isotopic weapon would be a nuclear/radiological incident.

b. Some of the most critical threats facing the US today are terrorists and organized crime groups. They are also among those most likely to use a RDD. An RDD, as defined by a 1979 US DOD report to a US/Soviet committee on disarmament, is *any device, including any weapon or equipment other than a nuclear explosive device, that is specifically designed to employ radioactive material by disseminating it to cause destruction, damage, fear, or injury by means of the radiation produced by the decay of such material*. Most RDD are used for the strong psychological impact they have on troops as well as the civilian population.

c. Typically, RDD are low-technology devices and may be as rudimentary as the abandonment of a radioactive source in a populated area. Medical sources, industrial irradiators, and radioactive waste can easily be obtained for use as simple dispersal devices. Conventional explosives can be used for the deliberate destruction of a nuclear reactor. Alternatively, reactors can be used to produce RDDs by production of specific radionuclides although this may require more complex technology and sophisticated techniques. Scale is the primary difference between contamination produced by RDDs and nuclear detonation. Another difference is that an RDD may contain highly concentrated sources that remain intact after the RDD explodes or otherwise comes apart. The probability of spreading contamination from encapsulated sources is greatly reduced. However, the emphasis should be placed on binding the contamination to an agent that will reduce re-suspension on isolated areas that have been contaminated. Overall, RDDs normally involve--

- Radioactive material combined perhaps with conventional high explosive.
- Medical and/or industrial isotopes.
- Cobalt, cesium, iodine, plutonium/ beryllium, and spent nuclear fuels.
- Unsophisticated delivery systems.
- Area denial weapons/psychological weapons.

d. If the terrorist goal is to contaminate reservoirs or other municipal water supplies, the fact that most radioactive material is not soluble in water means that its use by a terrorist would be unlikely and impractical. Also, the radioactive material will settle out or be trapped in filters. Those factors, coupled with the fact that any radioactive material will present safety risks to the terrorists themselves, collectively indicate the serious difficulties for any adversary attempting to store, handle, and disseminate it effectively.

e. In general, if the RDD involves fissile materials, such as the destruction of a nuclear reactor, the primary hazards are the same as for nuclear weapon fallout Initially, external whole-body gamma irradiation and beta burns will present the greatest threat. As time passes, the relative amount of short-lived fission fragments decreases due to decay. The primary hazard then becomes the internalization of alpha emitters, such as uranium and plutonium; however, external and whole-body hazards can remain due to long-lived beta and gamma emitters such as strontium and cobalt. This type of device is very effective at transforming standard injuries into combined injuries by radiological wound contamination. Also, there are no official casualty predictions for RDDs since the very nature of the weapon precludes verification of its actual use.

2-4. Nuclear Terrorism

a. Terrorist Groups. A terrorist group is defined as a collection of individuals belonging to an autonomous nonstate or subnational revolutionary or antigovernment movement who are dedicated to the use of violence to achieve their objectives. The reasons terrorists may perpetrate a weapons of mass destruction (WMD) attack include a desire to kill as many people as possible "to annihilate their enemies," to instill fear and panic to undermine a governmental regime, to create a means of negotiating from a position of unsurpassed strength, or to cause great social and economic impact. Such groups are seen as having at least some structure and command and control (C2) apparatus that, no matter how loose or flexible, provides an overall organizational framework and general strategic direction.

b. State-Sponsored Terrorism. State-sponsored terrorism is the active involvement of a foreign government in training, arming, and providing other logistical and intelligence assistance as well as sanctuary to an otherwise autonomous terrorist group for the purpose of carrying out violent acts on behalf of that government against its enemies. State-sponsored terrorism is, therefore, regarded as a form of surrogate warfare and is a critical aspect to be analyzed, since more than 20 countries are suspected of proliferating nuclear, biological, and chemical (NBC) weapons technology. However, several things work against state sponsorship in providing WMD to terrorist groups. These include the prospect of significant reprisals by the US against the state sponsor, the potential inability of the state sponsor to control its surrogate, and the prospect that the surrogate cannot be trusted; even to the point of using the weapon against its sponsor. It is significant that, to date, there is no evidence that any formal link exists between terrorist groups and state-assisted NBC programs.

c. *Terrorist Acquisition of Nuclear Weapons and Sensitive Nuclear Material.*

(1) *Nuclear weapons.* Terrorists who are able to acquire NBC weapons represent a major potential threat to the US' security and that of other nations. However, after the collapse of the Soviet Union, Western fears about security at Russian military facilities were heightened. It appears that Russian strategic and tactical nuclear weapons are perhaps more secure than had been initially feared. Where there may be particular concern is during their transportation for maintenance or dismantling, when the Russian weapons apparently are not subject to the same strict security measures. Even if terrorists were able to acquire a nuclear weapon, they would still face a number of significant obstacles in using or detonating it. Strategic nuclear warheads are immense and would be extremely difficult to move clandestinely without extensive preparation. Tactical nuclear weapons, such as artillery projectiles, are far lighter and easier to conceal, making them potentially much more attractive items for terrorist theft or illicit acquisition. Another obstacle is that many tactical nuclear weapons, and most strategic nuclear devices, are equipped with permissive action links or other security mechanisms such as tamper-proof seals which are designed to prevent accidental or unauthorized detonation. Therefore, it would be extremely difficult for terrorists to circumvent or overcome these built-in protective measures. Some of the smaller KGB (Komitet Gosudarstvennoy Bzopasnosti) suitcase weapons may have had little or no protective devices or locks installed and, thus, the safety measures designed to thwart unauthorized detonation would be more easily overcome.

(2) *Sensitive nuclear material.* Terrorists who were either unable or unwilling to steal a nuclear device, or were unsuccessful in obtaining one on the putative black market that has surfaced in the countries of the former Soviet Union and Warsaw Pact, might attempt to build one themselves. Their first hurdle, however, would be in acquiring sensitive nuclear material (SNM), that is, either highly enriched uranium (HEU) or plutonium suitable for fashioning a nuclear device. Mining and processing uranium or building a reactor to create plutonium would, of course, be impractical; terrorists would, therefore, have to steal SNM or conceivably purchase it on the black market. A number of authorities in recent years repeatedly have expressed concern about illicit access to nuclear materials and technology, particularly in the former Soviet Union. Minatom, the Russian entity with responsibility for nuclear weapons, has itself complained about a lack of qualified personnel and adequate control systems, and the lax security at HEU storage facilities. Given this apparent lack of security and the fact that 250 tons of HEU and 50 tons of weapons-grade plutonium has been stockpiled in Russia, the risk of illicit acquisition from SNM storage facilities should be considered a serious threat. Therefore, efforts directed at preventing the acquisition of fissile material are the first line of defense against nuclear terrorism.

(3) *The black market.* Potentially less worrying, however, is the supposed *black market* for these substances. In spite of the impression that organized crime groups are acquiring and selling NBC weapons, this is highly unlikely. It is important to make the distinction between what a group claims to have for sale and what it actually has in its possession. Between 1992 and 1996, more than 1,000 claims were made involving the illicit sale and smuggling of nuclear material. However, only six instances were substantiated, and none of those involved the quantities needed to construct an effective *homemade* device that could cause mass casualties--thereby suggesting that the nuclear black market, if it exists at all, is limited in size and grossly exaggerated in impact.

d. Challenges in Building a Device.

(1) Building a nuclear device capable of producing mass destruction presents Herculean challenges for terrorists and even for states with well-funded and sophisticated programs. According to one analysis, minimum requirements include *personnel, skills, information, money, facilities, equipment, supplies, security, special nuclear materials. . . and, usually, other specialized and hard-to-obtain material*. A successful program hinges on obtaining enough fissile material to form a supercritical mass for the nuclear weapon to permit a chain reaction. The weapon must then be designed so that it will bring that mass together in a tiny fraction of a second, before the heat from early fission blows the material apart. The device must also be small and light enough to be carried by a given delivery vehicle. It is important to emphasize that the above represents the *minimum* requirements. If each one is not met, the terrorist ends up with a device that cannot produce any significant nuclear yield at all, or cannot be delivered to a given target. Although it uses relatively scarce HEU, the gun-type device is considered technically easier to fabricate; and many analysts accordingly argue that terrorists attempting to make a bomb *in house* will build a gun-type device.

(2) There are different assessments about what level of expertise and other resources are required to construct such a weapon. According to one authority, *most states and some exceptionally capable nonstate actors* could build a highly destructive 10-KT weapon in several months at a cost of a few hundred thousand dollars, assuming they had access to sufficient quantities of fissile material. Other experts, however, are far more skeptical in their estimates of the capabilities required. Although much of the information about nuclear weapons design and production has become public knowledge during the past 50 years, it is still extraordinary for nonstate entities to attempt to embark on a nuclear weapons research and development program. Even technical requisite knowledge and hands-on experience are not enough to build an effective nuclear weapon. As an Office of Technology Assessment report explains, *[k]nowledge must be supplemented by industrial infrastructure and the resources to carry a nuclear weapon program to completion. The technologies for building cars and propeller-driven airplanes date back to early in this century, but many countries still cannot build them indigenously*.

(3) Any nuclear weapons program will, by nature, involve a number of people, and significant resources, equipment, and facilities. The ultimate irony is that all of this type of activity will significantly increase the risk of exposure of the terrorist group to detection by intelligence and law enforcement agencies. Therefore, theft of a nuclear device or building a weapon *in house* are the least probable courses of action for a prospective nuclear terrorist. Far more likely--for all the reasons cited above--is the dispersal of radiological material using an RDD (see paragraph 2-3).

e. Examples of Terrorist Activities. For many years it was generally believed that terrorist groups did not seek to kill large numbers of people at a time, but rather wished to demonstrate that they could execute attacks at will against civilian and military targets. In the wake of the use of Sarin gas in Tokyo, as well as the Oklahoma City, Pan Am, and Riyadh bombings, it is no longer possible to assume that genuine mass murder is not an intended

component of subnational forces, particularly if they are acting as state surrogates. Examples of terrorist acts that have escalated to the realm of mass murder are--

- The bombing of major US overseas targets, including the US Embassy and Marine barracks in Lebanon.
- The truck bombing of US forces' residences at the Khobar Towers in Riyadh, Saudi Arabia.
- Domestic incidents in Oklahoma City and at the World Trade Center.
- The explicit use of chemical warfare (CW) agents, as in Aum Shinrikyo's Sarin attack on the Tokyo subway system.
- The 1982 destruction of nonoperational reactors at two South African power stations by the terrorist wing of the African National Congress.

Also, in November 1995, in one of the few recorded incidents of specific nuclear terrorism, Chechen rebels placed Cesium-137 in a busy Moscow park. Although the material was packed in a protective canister, and thus posed no real threat, the incident embarrassed the Russian government, which was probably the Chechens' ultimate goal.

2-5. Regional Threats

a. General. Certain countries have embarked on extensive efforts to acquire and develop WMD (see Figure 2-1). The current world is multipolar, small-scale, and possibly nondeterrable, versus the previous situation where the two superpowers were predictable and deterrable. These countries develop WMD to--

- Attain and retain regional dominance.
- Attempt to negate the US military advantage.
- Threaten higher US casualties during potential conflicts.
- Complicate US military planning.
- Add to the perception of their military strength.
- Interfere with US buildup/early entry.
- Complicate operations by forcing protective measures.

Figure 2-1. Critical threat regions.

b. Dual Use Technology. To acquire the necessary technology, the buyer country or terrorist may use dummy corporations or a chain of purchasers to circumvent customs in the supplier country. The country or the group attempting to develop NBC weapons may also import a seemingly innocent dual use product; that is, one having both a benign use and a weapon use. Insulin is a medication for diabetics but also a catalyst for a biological warfare (BW) agent. Highly enriched uranium is a fuel for a civilian power reactor but also for a nuclear weapon.

c. North Korea. North Korea routinely engages in industrial and military espionage to gain technology. A Japan-based group also funnels advanced technology to the country. Although its nuclear program is expected to halt in the near future, North Korea will maintain and export its ballistic missiles and associated technology. North Korea also possesses tube and rocket artillery that ranges south of Seoul, while their missiles cover the entire region.

(1) *Nuclear program*. In the 1960s, North Korea obtained a Soviet research reactor and, in the 1980s, a plutonium-producing, nuclear fuel cycle, 5-megawatt (mW) reactor. Since that time, North Korea has produced enough plutonium for several nuclear weapons. Its fuel-reprocessing facility has been sealed and the International Atomic Energy Agency (IAEA) halted construction of 50 mW and 200 mW power reactors. North Korea has agreed to a framework treaty and obligations that include open inspection by IAEA and the eventual dismantling of all nuclear power facilities. However, according to a report in the Japanese business daily, *Nihon Keizai Shimbun*, the Japan's Defense Agency recently determined *there is a possibility North Korea may have at least one nuclear weapon*. It also stated that Pyongyang may have been driven to restart its nuclear weapons program by the Indian and Pakistani nuclear tests at the time. Despite fears that North Korea may obtain assistance for its nuclear program from Pakistan, there is not yet conclusive evidence of such technology transfer.

(2) *Biological program*. In the 1960s, North Korea began an offensive BW program. It probably can produce and weaponize limited quantities of traditional infectious agents or toxins.

(3) *Chemical program*. In the 1960s, North Korea began producing offensive chemical agents and, in the 1980s, expanded the program as part of its military preparedness plan. The program includes large quantities of nerve, blister, and blood agents. Since 1990, under North Korea's chemical defense readiness program involving the military and civilians, the entire population has been issued masks. Currently, the government is issuing antichemical gear to all military, including the reserves.

(4) *Delivery systems*. North Korea has SCUD-B (Western name for early Soviet missile series) (300 km) and SCUD-C (500 km) ballistic missiles and has acquired and tested the NODONG (1,000 km) and TAEPO DONG (4,000 km) ballistic missiles. In addition, the country has cruise missiles (100-km antiship), a wide variety of Soviet combat aircraft, and indigenous crop dusters and artillery.

d. Iraq. Iraq seeks to dominate the Persian Gulf by all means necessary. It has exhibited lack of cooperation with the United Nations and noncompliance with its Security Council resolutions. Its goal is to preserve as much of its NBC capabilities as possible through concealment, deceit, and intimidation.

(1) *Nuclear program*. Iraq received Osirak and Isis reactors from France in 1976 and has focused on producing highly enriched uranium. It is believed that Iraq could have a nuclear device within 5 years of the lifting of United Nations sanctions.

(2) *Biological program*. Iraq's extensive and aggressive biological program is believed to include 90,000 liters of botulinum toxin, 8,300 liters of anthrax, and significant quantities of carcinogens. Most of these munitions are weaponized. Although Iraq claims to have destroyed its biological agents, it could easily revive its BW program when inspections cease.

(3) *Chemical program*. Samarra is the location of a major chemical weapons production site. The production program, which is mostly self-sufficient, includes several thousand tons of blister and nerve agents. In the past, Iraq has used mustard gas and nerve gas on the Iranians. It is expected that, without United Nations sanctions, Iraq could revive its chemical weapons program within months.

(4) *Delivery systems*. Under the United Nations Security Council Resolution 687, Iraq is limited to 150-km range ballistic missiles but it has retained long-range missiles including the SCUD-B (300 km), Al Husayn (650 km), and Al Hijarah (650 km) missiles (see Figure 2-2). In addition, Iraq has the use of cruise missiles, rockets, combat aircraft and helicopters to deliver NBC weapons.

Figure 2-2. Range bands of Iraq's delivery systems.

2-6. Nuclear Weapons Employment

a. Tactical Nuclear Weapons. Combatants who possess a stockpile of tactical nuclear weapons must be assumed to have plans to use them within the constraints of their capabilities and the political environment. The weapons and yields discussed below are from open source data on US weapons. Potential threat nations are presumed to have a similar capability.

(1) Tactical nuclear weapons generally range in yield from 100 tons of TNT (2,4,6-trinitrotoluene) (0.1 KT) through 10 KT and even higher. The smallest tactical battlefield weapons deployed by the US were the 155-millimeter (mm) howitzer rounds known as artillery fired atomic projectile (AFAP). The first AFAP known was the M454 AFAP, which was reported to have a yield of 0.1 KT. This was later to be replaced with the XM785, which was to have had a yield of 0.2 KT. Theater-level weapons, such as the ground launched cruise missiles (GLCMs) and the Pershing II, which were deployed in Europe at the height of the Cold War, could have yields up to 400 KT. To place this in perspective, the bombs dropped on Hiroshima and Nagasaki were 20 KT and 17 KT respectively.

(2) Small-yield tactical nuclear weapons (delivered by tube artillery or medium battlefield rockets) would be planned for use against specific enemy units, key terrain on the battlefield, nuclear capable enemy units, or for shock value against specific troop concentrations. Generally, these would rarely exceed 10 KT. Larger yield theater weapons (for example, the Pershing II) would normally be used at the operational level against theater targets such as enemy long-range nuclear weapons systems, ports, airfields, and theater level logistic bases. Tactical nuclear weapons might also be deployed and utilized as a deterrence and response to either the use, or threat of use, of any WMD. This *right of first use* of nuclear weapons was the North Atlantic Treaty Organization (NATO) policy for the duration of the cold war.

(3) Large numbers of casualties with combined injuries would be generated within the lethal zone of the particular weapon(s) used. Casualties could also be produced at a later time due to fall-out. While large numbers of casualties would be generated, medical care

would be available outside the area of immediate destruction. The primary patient management concept would be to evacuate and distribute casualties to all available medical treatment facilities (MTFs).

b. Strategic Nuclear Weapons. Strategic nuclear weapons generally range from hundreds of KT to multiples of MT. They are designed to destroy large population centers, destroy or disrupt national and strategic nuclear forces and their C2, and to destroy or disrupt national infrastructure, logistics, and warfighting capabilities. The exchange of multiple strategic nuclear weapons would result in very large casualty numbers, which would overwhelm surviving local medical resources. Medical response for this occurrence is based on disaster mass casualty capabilities. Military personnel who are nominally capable of returning to short-term duty would be utilized despite significant radiation injury. Casualties would receive medical care and evacuation as available as soon as conditions permit. This threat is now considered the least likely potential nuclear scenario.

2-7. Casualty Estimates in a Tactical Nuclear Environment

a. General.

(1) This paragraph briefly discusses projected casualties due to limited use of nuclear weapons in a tactical environment. Depending upon the radiation protection available, nuclear detonations will generally produce initially a large number of blast, burn, and projectile injuries. The type of unit (heavy, light infantry, support) is key for the medical planner since the number of radiation and combined casualties is directly related to the protection available to that type of unit. The level of protection from radiation is expressed in terms of shielding. Material is available on the battlefield to construct/prepare expedient shelters that offer substantial shielding against gamma radiation. Of course, the armored fighting vehicles available to a heavy unit offer a distinct advantage in radiation protection, while retaining the ability to fight and maneuver. Generally, the denser or heavier the material, the better shielding it offers. The degree of protection afforded by shelters or vehicles is expressed as a *protection factor*, or a *transmission factor*. The transmission factor is the fraction (expressed in percentages or in decimals) of the available radiation dose which penetrates the material and reaches those inside compared to the radiation received by unprotected personnel. Thus, a transmission factor of 0.2 indicates that an individual within the shelter or vehicle receives two-tenths or 20 percent of the radiation dose he would receive if unprotected. Radiation transmission factors for some common shelters and armored vehicles are shown in Table 2-1.

*Table 2-1. Transmission Factors for Nuclear Radiation**

Environmental Shielding	Neutrons	Initial Gamma	Residual
Built-up City Area (in open)	1.0	0.5	0.7
Foxholes	0.3	0.2	0.1
Frame House			
First Floor	1.0	0.9	0.5
Basement	0.5	0.3	0.1
Multistory Buildings			
Top Floor	1.0	0.9	0.1
Intermed. Floors	0.9	0.9	0.02
Lower Floor	0.9	0.5	0.1
Basement	0.5	0.3	0.01
Closed Shelter, Earth Cover (3ft.)	0.05	0.02	0.005
Armored Vehicles			
APCs	0.3	0.2	0.1
Tanks	0.3	0.2	0.1
Wooded Forest	1.0	1.0	0.8

* Inside dose = Transmission factor times the outside dose.

It should be noted that the data for armored vehicles in Table 2-1 is for the M60 series tanks and M113 series armored personnel carriers (APCs). Therefore, one would expected that the M1A1 (heavy armor), the M1A2 Abrams tank series, and the Bradley fighting vehicle with much thicker and more dense armor would certainly transmit less than the percentages of external radiation as shown in the table.

(2) A detailed discussion of nuclear casualty estimates is available in Standardization Agreement (STANAG) 2475, *Medical Planning Guide of NBC Battle Casualties, Nuclear, AMedP-8 (A), Vol. 1*. Only a few examples are shown here to illustrate the effects of nuclear weapons depending upon yield and the type of unit. It should be noted that the casualty models used flat terrain, clear weather, and a low air burst to simulate the effects of 5 KT, 20 KT, and 50 KT weapons. Therefore, units occupying different terrain types or in cities could either have increased shielding or be more exposed depending upon circumstances. It should also be noted from the previous paragraph, that the use of tactical battlefield weapons above 10 KT would be rare, and, in fact, most tactical "strike packages" consist of weapons of much less yield than even 5 KT. Also note the tables showing casualties for a light infantry unit. Even when dug-in, the light infantry brigade is expected to suffer many more casualties in all areas of injury relative to a heavy unit. Therefore, with prior planning and training, forward medical units, especially those supporting heavy units, can successfully evacuate and treat soldiers with combined injuries and radiation injuries under most tactical circumstances.

b. In the example casualty tables (Tables 2-2 through 2-5), the following definitions are used for casualty causing effects:

- B-R-T: Casualties resulting from the combination of blast, radiation, and thermal.
- B-R: Casualties resulting from the combination of blast and radiation.
- B-T: Casualties resulting from the combination of blast and thermal.
- B: Casualties from blast alone.
- R-T: Casualties resulting from the combination of radiation and thermal.
- R: Casualties from radiation alone.
- T: Casualties from thermal alone.

The capable percentage ranges signify the number of personnel who are x percent capable after a nuclear strike. For example, **>25 percent-≤50 percent: 40**, means 40 personnel are projected to be more than 25 percent effective but less than or equal to 50 percent effective.

(1) Table 2-2 shows the status of unit personnel 1 hour after a nuclear strike; data were calculated by yield for a heavy brigade (4042 soldiers) in both an unwarned and a warned posture.

Table 2-2. Status of Unit Personnel by Yield; Heavy Brigade (4042 Soldiers)--Forward Maneuver--Movement to Contact--One Hour

		UNWARNED			**WARNED**		
		5 KT	**20 KT**	**50 KT**	**5 KT**	**20 KT**	**50 KT**
Casualties By Injury Type	B-R-T	45	16	0	8	2	2
	B-R	8	0	4	33	11	12
	B-T	0	0	9	0	0	0
	B	0	0	0	0	0	0
	R-T	0	0	0	0	0	0
	R	0	0	0	0	0	0
	T	0	0	0	0	0	0
Total Casualties		53	16	13	41	13	14
Fatalities		30	121	147	30	102	111
Total Capable		3959	3905	3882	3971	3927	3917
>25% - <=50%		40	26	40	33	14	20
>50% - <=75%		20	43	56	23	20	22
>75% - <100%		114	228	255	130	285	344
= 100%		3785	3608	3531	3785	3608	3531

(2) Table 2-3 shows the status of unit personnel of a heavy brigade 7 days after a nuclear strike. Note that, in general, the number of casualties and fatalities go up due to delayed effects of thermal and combined injury.

Table 2-3, Status of Unit Personnel by Yield; Heavy Brigade (4042 Soldiers)--Forward Maneuver--Movement to Contact--Day Seven.

		UNWARNED			WARNED		
		5 KT	**20 KT**	**50 KT**	**5 KT**	**20 KT**	**50 KT**
Casualties By Injury Type	B-R-T	9	0	0	8	2	0
	B-R	27	14	12	40	20	20
	B-T	0	4	4	0	0	0
	B	0	0	0	0	0	0
	R-T	6	0	0	2	0	0
	R	0	0	0	0	0	0
	T	4	14	47	0	2	6
Total Casualties		46	32	63	42	22	26
Fatalities		89	145	164	42	119	129
Total Capable		3907	3865	3815	3919	3901	3887
>25% - <=50%		0	0	0	0	0	0
>50% - <=75%		8	17	21	4	4	4
>75% - <100%		114	240	263	130	289	348
= 100%		3785	3608	3531	3785	3608	3531

(3) Table 2-4 shows the status of unit personnel of a light infantry brigade 1 hour after a nuclear strike. Note the significant rise in the number of fatalities and casualties as compared to a heavy brigade, even when elements of the infantry brigade are dug-in for the defense.

Table 2-4. Status of Unit Personnel by Yield; Light Infantry Brigade (3454 Soldiers)--Defense--One Hour.

		UNWARNED			WARNED		
		5 KT	**20 KT**	**50 KT**	**5 KT**	**20 KT**	**50 KT**
Casualties By Injury Type	B-R-T	459	842	688	242	450	472
	B-R	0	0	0	30	66	161
	B-T	0	0	276	0	0	120
	B	0	0	0	0	0	0
	R-T	0	0	0	0	0	0
	R	0	0	0	0	0	0
	T	0	0	0	0	0	0
Total Casualties		459	842	964	272	516	753

Fatalities	194	898	1802	248	689	1093
Total Capable	2801	1714	688	2934	2249	1608
>25% - <=50%	741	720	650	480	511	618
>50% - <=75%	374	832	38	436	746	294
>75% - <100%	1666	162	0	1998	992	696
= 100%	20	0	0	20	0	0

(4) Table 2-5 shows the status of unit personnel of a light infantry brigade 7 days after a nuclear strike. As in the heavy brigade, note the significant rise in the number of fatalities and casualties due primarily to the manifestation of injuries because of radiation and thermal effects.

Table 2-5, Status of Unit Personnel by Yield; Light Infantry Brigade (3454 Soldiers)--Defense--Day Seven.

		UNWARNED			**WARNED**		
		5 KT	**20 KT**	**50 KT**	**5 KT**	**20 KT**	**50 KT**
Casualties By Injury Type	B-R-T	196	103	0	135	123	111
	B-R	0	0	0	189	161	239
	B-T	0	0	99	0	0	37
	B	0	0	0	0	0	0
	R-T	679	139	0	342	68	0
	R	0	0	0	0	0	0
	T	0	809	542	0	406	283
Total Casualties		875	1051	641	666	758	670
Fatalities		789	1983	2803	603	1402	1945
Total Capable		1790	420	10	2185	1294	839
>25% - <=50%		0	0	0	10	0	0
>50% - <=75%		74	78	0	137	184	75
>75% - <100%		1696	342	10	2018	1110	764
= 100%		20	0	0	20	0	0

c. It must be emphasized that these tables are based on a standard computer model (Janus) used to simulate force-on-force engagements for training purposes. This model assesses the effects of conventional and NBC weapons as a function of time. Computer modeling is not perfect, and has limitations. However, the estimates shown above should give the planner an adequate baseline to begin a detailed analysis and to develop a good medical support plan.

Section II. NUCLEAR INCIDENTS AND LOW-LEVEL RADIATION

2-8. General.

a. From 1944 to 1997, radiation incidents throughout the world involved mainly radiation devices and isotopes. Accidents involving sealed sources of radiation accounted for about 50 percent of these incidents. Also, a few very serious incidents involving nuclear weapons have occurred throughout world. In addition to accidents, US forces have been recently deployed to areas where there is an increased risk of exposure to radiological, chemical, and biological contamination (Somalia and Eastern Europe, for example). US forces may be operating in a theater that has nuclear reactor(s) with varying degrees of safety and containment. Tactical dictates may require units to situate downwind of these reactors. The nuclear reactors and their containment facilities may be lucrative enemy artillery or bombing targets because of both the disruption of the electric power and the release of large quantities of radionuclides. The resources then diverted to crisis and consequence management of irradiated US personnel and host nation personnel may be overwhelming. Environmental radiological problems are of special concern since at very low levels of radiation there will not be any immediate outward signs of exposure. Other potential threats are those of industrial origins and radiological sources that may be used as booby traps or dispersed to enhance exposure (RDD). This section will discuss the most prevalent radiation sources that US forces could encounter because of an incident and/or an emission of low-level radiation. For a detailed discussion of these potential hazards, see Technical Guide (TG) 238, *Radiological Sources of Potential Exposure and/or Contamination*.

b. Crucial issues for a response force during a nuclear incident are health and safety concerns stemming from possible radioactive contamination, public affairs steps to address public concerns, security of classified information, and recovery of the weapon or containment of the radioactive source. Medical and environmental responders must identify the type of radiation emitted because of the accident, estimate dose-equivalent limits, begin remedial actions, initiate treatment, and institute countermeasures. In addition, planning must provide for medical assistance, security, logistics, legal advice, site restoration, communication, and response force integration. Intervention planning should also include countermeasures to limit acute radiation syndrome and other nonstochastic effects as well as efforts to reduce the collective dose so as to limit stochastic effects such as cancer induction and *in utero* exposures.

2-9. Natural Radioactive Sources

a. Background Radiation. Natural background radiation comes from the sun (cosmic radiation), radioactive elements in the earth's crust, rocks, air, water, and radioactive materials in plants and the human body. Background radiation is unavoidable and the amount varies from one location to another. Examples of background radiation are included in Table 2-6.

Table 2-6. Examples of Background Radiation

Source	Radiation Type or Radionuclide
Cosmic Radiation	Gamma, Electrons, Protons, Neutrons, and Cosmic Rays
Earth's Crust	U, Th, ^{40}K, Natural Decay Elements, and Others
Air	Radon (^{222}Rn), Thoron (^{220}Rn), ^{3}H, ^{14}C, and Others
Water	U, Th, Ra, ^{40}K and Others
Plants and the Human Body	^{226}Ra, ^{228}Ra, ^{3}H, ^{14}C, ^{40}K, and Others

b. Cosmic Rays. Prior to entering the earth's atmosphere, cosmic rays mainly consist of protons and alpha particles. Upon interaction of cosmic rays with atoms in the atmosphere, electrons, photons, protons, and neutrons are created. Carbon-14 (^{14}C) and tritium (^{3}H) are also created by interactions of cosmic rays with the atmosphere. Ninety percent of the tritium from cosmic rays is found in the ocean and other terrestrial waters. The remaining 10 percent are found in the stratosphere, where the tritium is directly produced by the cosmic ray reactions.

c. Earth's Crust. Soil contains radioactive materials such as uranium, radium, thorium, potassium 40, and other elements. Some radioactive elements found in soil decay to become part of the natural radiation decay series. For example, radon (^{222}Rn) and thoron (^{220}Rn) are part of the decay process in the uranium and thorium series. Uranium is present in virtually all rocks and soils, as well as in rivers and seawater. It is also associated with three of the four naturally occurring radiation decay series. Uranium's overall concentration in the earth's crust is around two parts per million (ppm). This equates roughly to 1 pound of uranium in every 500,000 pounds of earth. In its natural state, uranium is a slightly radioactive heavy metal and is in an oxide form. Pure refined uranium metal is one of the most dense metals; it is about 65 percent more dense than lead. Hence, one of its military uses is as the depleted uranium (DU) solid rod penetrator for the US 120-mm hypervelocity discarding sabot kinetic energy antitank round (see paragraph 2-14). Radium 226 is also found in the soil, and naturally decays to Radon 222. Radon 222 is a radioactive gas that diffuses out of the ground and into the atmosphere, and therefore, provides a large contribution to environmental radioactivity.

d. Air. Radon and thoron are radioactive gases that become airborne as they leak out from the soil or building materials. Therefore, the amount of these gases present in the atmosphere depends on the amount of the radioactive material present in the soil, the porosity of the surface materials, ventilation, and meteorological conditions. Buildings without proper ventilation can trap radon inside where it may accumulate to a point where it becomes a health hazard. When one breathes air containing a large amount of radon, the radiation from its progeny due to natural decay can damage the lungs. As mentioned before, Carbon-14 and tritium are also present in the air because of interaction with cosmic rays.

e. Water. The radioactive materials found in water depend upon the source of the water. For example, well water may contain traces of uranium, thorium, radium, and it is often high in radon; whereas, seawater may have higher concentrations of ^{40}K. The tritium created in the atmosphere by interaction with cosmic rays gradually descends into the lower part of the atmosphere by natural diffusion. It is then brought down as tritiated water by rain or snow to the earth's surface, and accumulates in terrestrial waters. The testing of thermonuclear weapons in the atmosphere brought an increase in the amount of tritium on the earth; however, tritium is fairly uniformly distributed wherever water is present.

f. The Human Body and Plants. The human body and plants have radioactive materials that have been transferred by the food chain. For example, bones contain radioactive radium and other "bone seeker" radioisotopes. A typical human body contains about 100-125 micrograms of uranium. The daily uranium intake (largely from food ingestion) and excretion is about 1.9 micrograms. Bone contains a few thousand picograms (pg) of uranium per gram of bone ash; however, soft tissue concentrations are relatively low, in the order of a few hundred pg per gram of wet tissue.

g. Average Yearly Dose. Every person on earth is exposed to ionizing radiation every day. The average US resident receives a dose of about 360 millirem (mrem) per year. The most significant contributor to this dose is radon, a gas created by the radioactive decay of uranium. Since uranium occurs naturally in the ground and rocks, including building materials, radon seeps into buildings where it concentrates in enclosed spaces and is inhaled. The exposure rates in Figure 2-3 are for the average person. Note that the average person receives less than one mrem per year from nuclear power production and fallout from atmospheric weapons testing. The annual radiation dose that each person receives also varies by geographic location. For example, the background radiation levels in Denver, Colorado are higher than those at sea level, since it has less air between it and the sun than does a city at sea level. Air, although not dense, still acts as a shield from cosmic rays.

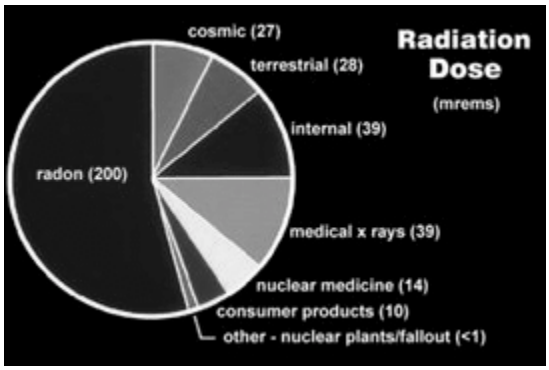

Figure 2-3. Yearly dose.

2-10. Sources from Nuclear Reactors and the Nuclear Fuel Cycle

a. Nuclear Reactors. As of 1999, there were 494 nuclear power plants worldwide; 444 were in operation in 1997, decreasing to 433 in operation in 1999. It is important to understand the basic types, components, and waste products of reactors to understand the types of radioactive exposure which might occur due to a nuclear reactor accident. Shown in Table 2-7 is the worldwide distribution of nuclear plants by reactor type.

Table 2-7. Types of Nuclear Reactors

Reactor Type	**Percentage**
Pressurized Water Reactor (PWR)	58
Boiling Water Reactor (BWR)	20
Gas Cooled Reactor (GCR)	7
Heavy Water Reactor (HWR)	11
Light Water Graphite Reactor (LWGR)	3
Fast Breeder Reactor (FBR)	1

Typical components of nuclear power reactors are discussed below:

(1) *Reactor pressure vessel*. In most reactors, the fuel is contained within a single pressure vessel. However, in some designs, the fuel rods are contained within individual pressure tubes.

(2) *Fuel*. The fuel is usually an arrangement of pellets of enriched uranium oxide (UO_2) in zircalloy rods, which are placed in the reactor core. Most commercial reactors use UO_2 as fuel, although some breeder reactor and gas cooled reactor designs may use plutonium oxide (PO_2) and thorium oxide (ThO_2) as fuel.

(3) *Moderator*. The moderator is used to slow down the fast moving neutrons produced by fission. This allows them to be captured by other nuclei so that the fission process continues. The moderator may be in the form of a solid, such as graphite, or a liquid, such as water.

(4) *Control rods*. Control rods are made of neutron absorbing materials such as cadmium or boron and are strengthened with steel. They are moved into or out of the reactor core to control the rate of fission or to stop the reaction completely.

(5) *Coolant*. The coolant is a liquid or a gas circulating through the reactor, transferring heat away from the core generally in the form of steam; see (6) below.

(6) *Steam generator*. The steam generator is part of the cooling system of a PWR. It separates steam from the coolant and directs it to the turbines to generate electricity.

(7) *Containment*. Reactors are contained inside a casing that acts as a radiation shield and is designed to prevent the release of radioactivity into the environment. Lack of adequate containment is a design weakness of some former Soviet Union designed reactors.

(8) *Other Components*. Other important components include the reactor pumps, turbines, pressurizer (in PWRs), and demineralizers.

b. Waste from Nuclear Reactors. The PWR is the most common type of nuclear power plant in the world. Therefore, its waste has become the most common type of radioactive waste from nuclear power plants. This waste is generated from liquid, solid, and gaseous effluents. Most facilities classify their waste as clean, dirty, and laundry wastes. The terms clean and dirty refer to the chemical purity of the water and not to the amounts of radioactivity. For example, the primary system waste that originates in the primary coolant circuit is essentially chemically pure water, but has the highest level of radioactivity.

(1) *Liquid waste*. The presence of radioactive materials in the reactor coolant system (RCS) is a result of neutron activation of chemicals, impurities, and the water contained in the system, and from leakage due to defects in the reactor fuel rods. The radioactive material is transferred from the fuel to other parts of the plant via the RCS, coolant cleanup systems, heat exchangers, and so forth. A holdup tank is used to collect the liquid for about 30 days in order to allow the decay of radionuclides with a short half-life. Radioactive liquid waste is then decontaminated by evaporation or demineralization.

(a) In the evaporation process, water is boiled off and the steam is condensed. The evaporator residue (solids) contains most of the radioactive material and can be disposed in a controlled manner. The condensed water is essentially, although not completely, free of dissolved solids, but will contain all of the tritium. The tritiated water is vaporized and then condensed with the ordinary water in the evaporator.

(b) In the demineralization process, an ion exchange resin is used which is similar to the material used in household and industrial water softeners. Most of the dissolved matter is removed and retained by the resin when the radioactive wastewater is passed through the demineralizer. Elements such as cesium, yttrium, and molybdenum are removed relatively slowly by demineralizers, and tritium is essentially unaffected.

(2) *Gaseous waste*. Gaseous effluents from power plants contain noble gases such as krypton and xenon, iodine, and particulate matter. Particulate matter is reduced by filtering the gas through a high efficiency particle arrestor (HEPA) filter prior to discharge. A charcoal filter removes part of the iodine. The noble gases are difficult to remove, hence they are held for a time to permit radioactive decay. The gasses are compressed and are held for about 60 days. Krypton-85 (^{85}Kr), ^{133}Xe, ^{133}I, and tritium are the radionuclides that remain in appreciable amount. These gasses are filtered and mixed with large volumes of ventilation air prior to discharge through the stack.

(3) *Solid Waste*. Solid waste actually includes the spent fuel and reactor components themselves. Aside from these items, solid waste generally includes the spent ion exchange (demineralizer) resins, discarded filter material (including charcoal), and evaporator residues. The residues are often slurries rather than solids, but are usually solidified by mixing with cement.

c. Radiation Exposure Pathways. Table 2-8 shows the principal radiation exposure pathways from nuclear power plant effluents. Because of their relatively large yield in uranium fission and known affinity to be deposited in the thyroid (a critical organ), iodine-131 and iodine-133 are very important sources of exposure. Cesium-134 and Cesium-137 are the radioisotopes produced in significant amounts during fission, and, if ingested, exposes all organs to beta-gamma radiation. Cesium can enter the body by drinking water or milk, or by eating fish from the general vicinity of the liquid effluent discharged from the plant. The internal (lung) dose from inhalation of radioactive isotopes of noble gases is very small. If adequate holdup is provided before release into the atmosphere, exposure to short-lived noble gases, such as ^{133}Xe and ^{88}Kr can be minimized. However, there may be significant exposure to long-lived ^{85}Kr if it is allowed to accumulate in the atmosphere. Tritium may also present an exposure risk if allowed to accumulate in the liquid and gaseous effluents and in the surrounding environment.

Table 2-8. Radiation Exposure Pathways from Nuclear Plants.

Radiation From	**Effluent**	**Exposure Pathway**	**Critical Organ**
Iodine Isotopes	Airborne	Ground Deposition (external) Air Inhalation Grass-to-Cow-to-Milk Leafy Vegetables	Whole Body Thyroid Thyroid Thyroid
Iodine Isotopes	Liquid	Drinking Water Fish (Shellfish Consumption)	Thyroid Thyroid
Tritium	Airborne	Submersion (external) Air Inhalation	Skin Whole Body
Tritium	Liquid	Drinking Water Food Consumption	Whole Body Whole Body
Cesium Isotopes	Airborne	Ground Deposition (external) Grass-to-Cow-to-Milk Grass-to-Cattle-to-Meat Inhalation	Whole Body Whole Body Whole Body Whole Body
Cesium Isotopes	Liquid	Sediments (external) Drinking Water Fish Consumption	Whole Body Whole Body Whole Body
Isotopes of Metals (iron, cobalt, nickel,	Liquid	Drinking Water Fish Consumption	GI Tract GI Tract

zinc, manganese)			
Direct Radiation from the Plant		External	Whole Body

d. Waste from Nuclear Fuel Cycle.

(1) The nuclear fuel cycle includes all the activities associated with the production of electricity from nuclear reactions. This includes mining of the fuel, milling, conversion, enrichment, fuel fabrication, the reaction, and the resultant spent fuel. Waste from the nuclear fuel cycle is categorized in a number of ways, including high, medium, or low-level waste. These wastes come from virtually all phases of the cycle and include--

- Essentially nonradioactive waste resulting from mining.
- Low-level waste produced at all stages of the fuel cycle.
- Intermediate-level waste produced during reactor operation and by reprocessing.
- High-level waste, which is spent fuel and waste containing fission products from reprocessing.

(2) Milling waste contains long-lived radioactive materials and progeny in low concentrations and toxic materials such as heavy metals; however, the total mass activity of radioactive elements in milling waste is less than the original ore. This waste requires safe management in order to isolate it from the environment or to ensure that releases are limited to reduce environmental impact. Normally, shallow burial in engineered facilities is used to dispose of milling waste.

(3) The chemical conversion process of turning uranium hexafluoride into dioxide produces liquid waste that contains chemical impurities, including fluorides. Lime is used to treat the effluent in order to precipitate calcium fluoride that contains traces of uranium and some decay products.

(4) The fuel enrichment process leads to the production of depleted uranium (DU). This is uranium in which the concentration of ^{235}U (radioactive isotope) is significantly less than the 0.7 percent found in nature. This material, which is primarily ^{238}U (non-radioactive isotope), is used in applications where a high density material is required. Some of the applications include radiation shielding, production of mixed oxide (MOX) fuels, main battle tank armor, and kinetic energy armor piercing rounds (see paragraph 2-14). While ^{238}U is not fissionable, it is a low specific activity radioactive material and some precautions must be taken in its storage and disposal.

2-11. Biomedical Sources

a. Sources of Biomedical Radiation. Biomedical sources of radiation are those devices or materials that are readily available at hospitals and some laboratories, which could present a hazard if individuals are exposed or if the materials are dispersed into the surrounding environment. An example would be the intentional release of radionuclides into the environment from a nuclear medicine department due to a terrorist-caused explosion and fire. Biomedical uses of radiation are roughly broken down into two areas--diagnosis and therapy. Diagnosis includes routine x-rays, fluoroscopic examinations, computed tomography exams, and exams performed in nuclear medicine such as injections of radioactive materials. Therapy is primarily treatment for cancer, and includes radiation-producing equipment such as linear accelerators, radioisotope-generating machines, and radiation from brachytherapy sources.

b. Diagnostic Radiation Sources. For all of the devices discussed below, accidents can be precluded by simply having the devices turned off. However, during operation, the three basic ways to reduce external doses can be applied (minimize time and maximize distance and shielding). The following are common radiation-producing diagnostic devices and materials:

(1) *X-ray units*. X-ray radiation is produced by accelerating electrons through an electrical voltage potential. As soon as the switch is released, or the pre-set exposure time is reached, x-ray production ends. Many x-ray-type devices that use a high voltage and a source of electrons produce x-rays as an unwanted byproduct of device operation. These are called incidental x-rays.

(2) *Computed tomography*. Computed tomography *(CT)* is a diagnostic procedure in which a cross-sectional picture of a *slice* of the body is made. Sometimes a contrast material such as iodine is injected into the patient's vein. Modern CT scanners use narrow x-ray beams that concentrate directly on the area or organs under study. Computed tomography scanners do not present a hazard unless they are turned on and nearby personnel do not take routine shielding and distance precautions.

(3) *Fluoroscopy*. Fluoroscopy combines an x-ray unit and an image intensifier that allows a *real time* view on a screen of the patient and any procedures that are being performed on the patient. An x-ray beam passes through the patient through a series of intensifier tubes, fluorescent screens, and photocathodes. This produces light photons, that in turn, produce an image on an output screen. As with other x-ray devices, fluoroscopy units are not a danger unless they are in operation and personnel are within the beam field.

(4) *Diagnostic nuclear medicine*. This branch of medicine uses a radiation source positioned in the body to image a person's anatomy and to provide information about the functioning of a specific organ(s). Radiopharmaceutical tracers are attracted to specific organs, bones, or tissues. By putting a radioactive isotope in the body, its path through the body can be tracked by an external monitor. A special type of camera is used, a gamma or positron emission tomography (PET), or a single photon emission computed tomography (SPECT) camera. These cameras transform the emissions into images and data on the particular organ or process being imaged.

c. Therapeutic Radiation Sources. Radiation therapy departments treat patients for cancer or certain benign conditions. The most common types of therapy equipment are cobalt teletherapy equipment and linear accelerators. Rapidly dividing cells are particularly sensitive to damage by radiation. Therefore, some cancerous growths can be controlled or eliminated by irradiating the area containing the growth. Some cancers are treated with sealed radioactive sources placed directly in the tissue, the body cavity, or on the body surfaces. This type of treatment is called brachytherapy.

(1) *The cobalt-60 unit*. Of all potential radionuclides, ^{60}Co has proved to be the most suitable and most commonly used for external beam radiotherapy. The ^{60}Co source is usually in the form of a solid cylinder, disc, or pallet and is contained inside a stainless steel capsule and sealed by welding. The ^{60}Co decays to ^{60}Ni with the emission of beta particles and two photons of energy; these gamma rays constitute the useful treatment beam. Caution must be taken when handling teletherapy units because sources in the units may not be in the shielded position and therefore may be a serious radiological hazard.

(2) *The gamma knife*. The gamma knife delivers a single, high dose of ionizing radiation by sending beams from 201 separate ^{60}Co sources through a device known as the collimator helmet. Only at the point where all 201 beams converge at a single, finely focused point is enough radiation delivered to treat the diseased tissue. However, nearby tissue is spared. The gamma knife can present a radiological hazard if the shielding mechanisms fail, or if the ^{60}Co source unit is involved in an explosion or fire.

(3) *Heavy particle beam generators*. Heavy particle beam generators, cyclotrons, and linear accelerators produce heavy particle beams which include neutrons, protons, deuterons, alpha particles, and so forth, accelerated to high energies. Their use in radiotherapy is still experimental; however, they are undergoing clinical trials, and there is a certain amount of risk involved with this type of equipment.

(4) *Brachytherapy*. Brachytherapy is a method of treatment in which sealed radioactive sources are used to deliver radiation at a short distance by interstitial (implants), intracavity, or surface application. A high radiation dose can be delivered locally to the tumor with rapid dose fall off in the surrounding tissue. The radioactive sources are fabricated in the form of needles, wires, or seeds, which can be inserted into the tissue. These sources need to be handled in accordance with the proper safety precautions to preclude any unnecessary dangers. Table 2-9 shows some of the common radionuclides used in brachytherapy and their half-life.

Table 2-9. Radionuclides used in Brachytherap.

Radionuclide	**Half-Life**
Cobalt-60	5.26 Years
Palladium-103	17.0 Days
Iodine-125	60.2 Days
Cesium-137	30.0 Years
Iridium-192	74.2 Days

Gold-198	2.7 Days
Radon-222	3.83 Days
Radium-226	1600 Years

2-12. Industrial Sources

United States personnel can also be exposed to radiation from industrial processes involved with agricultural, scientific research, manufacturing, and educational facilities. Exposure from these sources may not be common, but accidental exposures can result in serious consequences. The use of radiation and radioactive materials can be found in many sectors of industry and science. Under normal operating conditions, most industrial sources of radiation present minimal exposure risks when used safely. However, US personnel must always be aware of their surroundings and be alert to the possibility of danger from these sources. Table 2-10 summarizes some of the industrial sources of radiation. Also, some of the more common sources are discussed in detail.

Table 2-10. Industrial Sources of Radiation

Locations and Materials	**Radiation Sources**	**Source Strength**	**Comments**
Gauges, Sources, Static Eliminators.	Iridium-192, Cesium-137, Cobalt-60, Radium-226, Neutrons, Americium-241, Polonium-210.	Greater than about 4 TBq.	Sealed sources, and if leaking, presents surface contamination.
X-ray Machine Sterilizers, Processors, and Particle Accelerators.	X-rays, Protons, Deuterons, Electrons, Gammas, Cesium-137, Cobalt-60.	~4 TBq to ~40 PBq.	Anywhere in an industrial area. Be aware of possible activation products.
Mineral Extraction and Processing, including phosphate fertilizers, oil, natural gas, and coal.	Naturally occurring Radioactive Materials-Uranium, Thorium, and their progeny.	Generally low level with external exposures from background level to about 0.01 mSv (1 mrem).	Dispersed low level material and scale build-up in piping. Also, in gauges as noted above. Radon is a possible concern.
Power Sources.	Plutonium-238, Strontium-90.	Plutonium-238: Up to 4 GBq; Strontium-90: Up to 1 TBq.	In equipment in isolated areas.
Radioluminescent Materials.	Promethium-147, Tritium, Radium-226.	Up to tens of TBq.	Various applications, and if leaking, surface contamination.

a. Radiographic Sources. Radiography is a technique that allows critical components of systems or machinery to be inspected for internal defects without damaging or disassembling the system. This form of inspection is sometimes referred to as nondestructive inspection (NDI). For example, radiography can be used for checking welds in pipeline joints or for finding flaws in metal castings. Typically, a small gamma ray source is sealed in a titanium capsule. This source is placed on one side of the object being screened and some photographic film is placed on the other side. The gamma rays pass through the object and crate an image on the film. Just as x-rays show a break in a bone, the gamma rays show flaws or breaks in the object being screened. Radiography techniques have the advantages over x-ray machines in that they can be taken to the work site, and no electrical power is needed. Examples of radionuclides used in industrial radiography are shown in Table 2-11.

Table 2-11. Radionuclides Used in Radiography

Radionuclide	**Gamma Energies (MeV)**	**Optimum Steel Thickness (mm)**
^{60}Co	High (1.17 to 1.33)	50-150
^{137}Cs	High (0.662)	50-100
^{192}Ir	Medium (0.2-1.4)	10-70
^{169}Yb	Low (0.008-0.31)	2.5-15
^{170}Tm	Low (0.08)	2.5-12.5

Potential hazards from these sources include--

- Inadequate monitoring of the intensity and duration of exposure, and not maintaining proper safety precautions.

- Compromised containers or improper methods of exposure resulting in the source remaining exposed.

- Improper long term storage procedures that expose personnel to higher dose rates.

b. Sterilization and Food Processing Sources. Radiation devices and radioisotopes are also used extensively in the processing of food. These devices are used for determining proper concentration of solutions (density gauges) and for sterilization-preservation (food irradiators).

(1) *Density gauges*. Density gauges read the amount of radiation as it passes through matter to measure the amount of attenuation or reduction in intensity. This measurement allows the user to detect the presence or absence or to judge the quantity of a material. Gauges are used in the food industry for measurement of sugar solutions, evaporated milk, chocolate, and soup seasoning. (Gauges can also be used in the chemical industry to determine the concentration of acid, alkali, saline solutions, and so forth.) In most devices, a ^{137}Cs source is

used that is double encapsulated, welded, stainless steel. The shielding container consists of a cast iron housing filled with lead. Potential hazards from this device would arise only if the shielding container and/or the source capsule is damaged, develops leaks, or is destroyed.

(2) *Sterilization*. Gamma irradiation is widely used for sterilizing not only food, but also for medical products and wool. Food preservation is an increasingly important application and has been used since the 1960s. In 1997, the irradiation of red meat was approved in the US. Some 40 countries, excluding Australia, have approved the irradiation of more than 50 different foods to extend the shelf life and to reduce the risk of foodborne diseases. The most common food irradiators use ^{60}Co and ^{137}Cs. A food irradiation facility resembles any other warehouse. Workers would typically load untreated food into containers attached to a conveyor system. The containers would then move into the irradiation chamber, which is commonly enclosed by thick concrete walls. Inside the irradiator, racks of pencil-shaped rods, which contain the radioactive material, rise from a pool of protective water to emit gamma radiation. Depending on the dose, these rays can retard spoilage, kill insects, or destroy bacteria. Potential hazards are if the radiation sources are exposed or dispersed. Personnel should adhere to a standard radiation safety program, including using appropriate radiation dosimetry and monitoring regimens.

2-13. Sources from United States Forces Commodities and Foreign Material

a. United States forces use many radioactive commodities in equipment, vehicles, ships, aircraft, weapons systems, and so forth. Rather than discuss every radioactive source available in US military equipment, this paragraph will discuss specific examples of the most common radioactive sources. Included will be a brief hazard assessment of each commodity.

(1) *Tritium (^{3}H).* Tritium is the heaviest isotope of hydrogen and the only one that is radioactive. Tritium is generally used in devices requiring a light source. Examples include watches, compasses, and fire control devices for tanks, mortars, howitzers, and so forth. The tritium is encased in a glass tube that has been lined with phosphor. The interaction between emitted beta particles and the phosphor produces visible light. Fire control devices that contain the most tritium are collimators and aiming lights, since they must be visible from several meters away. Tritium is a low energy beta emitter that cannot penetrate an intact glass tube. However, if the tube is broken, the tritium gas will dissipate and the outer surfaces of the device and in the vicinity of the break may become contaminated. Because of the weak beta radiation, tritium is not readily measured by the ordinary Geiger-Mueller counter, and it may require wipe testing to determine the level of contamination. Tritium is not absorbed by the skin to any significant degree. The hazardous nature of tritium is that when it is combined with water, it forms water vapor that is readily absorbed by the body (tritium oxide), both through inhalation and absorption through the skin. In turn, the radioactive water is distributed throughout body tissues.

(2) *Nickel-63.* Nickel-63 (^{63}Ni) is a pure beta emitter with a radiological half-life of 92 years, and is used in the chemical agent monitor (CAM). The ^{63}Ni is used to ionize air molecules as they pass through the detector. The beta energy of ^{63}Ni is too low to penetrate the

dead layer of skin; however, efforts should be taken to prevent ingestion, inhalation, or absorption through broken skin.

(3) *Cesium-137.* Cesium-137 (^{137}Cs) is used in the soil density and moisture tester (Campbell Pacific Model MC-1). It has a 10 mCi ^{137}Cs source located in a probe tip used to determine the density of the soil at a construction site. The ^{137}Cs emits a negative beta particle as it decays to ^{137}Ba, which in turn decays by emitting gamma rays. The hazard is minimal since the radioactive source is totally shielded in double encapsulated stainless steel.

(4) *Promethium-147.* Promethium-147 (^{147}Pm) has a half-life of 2.64 years and is a beta particle emitter used in luminous paints. The only weapon system that uses this element is the M72 series, 66 mm, light antitank weapon (LAW). For over 30 years, the LAW was manufactured with ^{147}Pm. Promethium-147 is located on the 100 m and 150 m range markers on the sight. In the early 1980's, the "E" version of the LAW's sight was changed, eliminating the need for ^{147}Pm. However, the older models of the LAW, M72, M72A1, and M72A2, still remain in use for training and are therefore still in long-term storage. There are no known hazards involved with ^{147}Pm.

(5) *Radium-226.* Radium-226 (^{226}Ra) was used on the faces and pointers of dials and gauges of instruments of tactical combat vehicles. Radium dials and radium marks on toggle switches may also be found in some radios of the older VRC-12 series, including the AN/GRC-19, the VRC-46, and the GRC-106. Radium-226 is primarily an alpha emitter, but it also emits some low energy beta and gamma radiation. The hazard was such that these commodities are no longer in use nor in DOD storage depots, and they have not been procured since 1969. Nonradioactive replacements have been available in the supply system for all of these radioactive items.

(6) *Thorium-232.* Thorium-232 (^{232}Th) is a naturally occurring radioisotope of thorium and is an alpha emitter. When thorium is heated in air, it glows with a white light. For this reason, one of the major uses of thorium has been the Welsback lantern mantle used in portable gas lanterns. Thorium-232 is also in radiac sets AN/VDR-2, AN/PDR-54, and the AN/PDR-77. Thermal optic fire control systems have a multilayer, infrared, antireflective coating that contains ^{232}Th as a fluoride compound (thorium fluoride). Thorium-coated optics are found on many night vision devices, such as the AN/TAS-4 series. Also, natural thorium oxide evenly dispersed in simple nickel thorium alloy is used in the combustor liner for the Abrams series tank turbine engine because the alloy can withstand a great amount of heat. In general, ^{232}Th presents a minimal hazard, but care should be taken to avoid inhalation or ingestion of any particles from damaged components that contain ^{232}Th.

(7) *Americium-241.* Americium-241 (^{241}Am) does not occur naturally; it is a daughter product of the decay of ^{241}P and has a half-life of 458 years. Americium-241 is used as a sealed source in the M43A1 Chemical Agent Detector that is a component of the M8A1 alarm. Americium-241 is primarily an alpha and a very low energy gamma emitter. External exposure is not a concern unless large amounts of the ^{241}Am are located in one area and personnel are in close contact for an extended period of time. The high energy alpha emission can present an internal radiation hazard if ingested. Americium-241 is chemically analogous to calcium and it can replace

calcium in the body, especially in bone. Therefore, as a "bone seeker," if ingested, it will be incorporated in the bone and surrounding tissue. This constant irradiation of bone tissue may potentially result in leukemia and malignancies.

b. Radioactive Sources in Foreign Materiel. Similar to US forces commodities, some foreign materiel contains radioactive sources. Although these sources do not present a hazard to personnel working close to them, it is important to be aware of their presence as they could be dangerous if the equipment has been damaged or tampered with. See AST-1500Z-100-93 and TG 238 for detailed descriptions of radioactive sources in foreign materiel.

(1) Various instruments and detectors, such as chemical detectors, icing monitors, and smoke detectors, use alpha sources. The most common are ^{241}Am, ^{226}Ra, and ^{239}P. These sources' primary hazard is the potential for internal radiation exposure due to contamination from their rupture, since these materials can be ingested or inhaled.

(2) Gauges, nuclear batteries, static eliminators, luminous dials, and other devices use beta sources. The most common beta sources used are ^{3}H, ^{90}Sr, and ^{147}Pm. The primary hazards associated with these sources are skin contamination and internal exposure if ingested or inhaled.

(3) Some foreign radiological test and measurement equipment use gamma sources. The most common gamma sources are ^{60}Co, ^{137}Cs, and ^{226}Ra. The amount of shielding required from these types of sources depends on their strength and the energy of the gamma rays produced during radioactive decay. Gamma sources represent both an external and an internal radiation hazard.

2-14. Depleted Uranium Armor and Munitions

Natural uranium is predominantly ^{238}U by weight, but also contains isotopes ^{234}U and ^{235}U. As part of the nuclear fuel cycle, natural uranium is processed in enrichment facilities to obtain uranium with a higher ^{235}U content; that is, enriched uranium. The enriched uranium is then used in nuclear reactors and nuclear weapons. The waste product of the enrichment process is uranium that has a lower content of ^{235}U and is known as DU because it is *depleted* of the radioactive isotope; it is, therefore, less radioactive than natural or enriched uranium. Table 2-12 shows a comparison between DU and natural uranium. The radiation emitted from DU is alpha, beta, and gamma, but because of the long half-life of ^{238}U, the specific activity is relatively low. For example, to obtain one curie of radioactivity from DU would require a single piece weighing 6,615 pounds.

Table 2-12. Comparison Between Depleted Uranium and Natural Uranium

Uranium Isotope	Natural Uranium	Depleted Uranium
^{234}U	0.0057%	0.0005%
^{235}U	0.7204%	0.2500%

^{238}U	99.2739%	99.7495%
Total	100%	100%

a. Because of its high density and structural properties, DU is useful for nonnuclear applications. One of these uses is that it can be applied defensively to protect against penetration by projectiles made of less dense metals, such as conventional solid rod kinetic energy tungsten carbide penetrators. The Abrams tank family (M1, IPM1, M1A1, and M1A2) has an improved hull armor envelope that does not contain DU. However, M1A1 heavy armor (HA) and M1A2s have DU "add-on" armor modules molded onto the existing left and right frontal turret armor. The front slope of the turrets of these models has a radioactive signature; a little less than 0.5 mrem/hour.

b. Depleted uranium is also used offensively in ammunition used to defeat enemy armored targets. The combination of high hardness, strength, and density makes DU alloys well suited for kinetic energy ammunition. Another useful property of DU is that as it moves through the armor it maintains the sharpness of the penetrator, further enhancing its penetrating power. (The fact that tungsten carbide penetrators do not sharpen on impact--but in fact mushroom to a certain extent--is one reason they are less effective for overcoming armor plating.) Current US weapons systems that can fire DU munitions are Abrams tanks, Bradley fighting vehicles, Air Force A-10 aircraft, Marine Corps Harrier aircraft, and the Navy's ship-mounted Phalanx Close-In Weapon System (CIWS). In general, DU ammunition may only be fired during actual combat, and is not fired in training situations. Peacetime firings on ranges are prohibited except on ranges which have been approved and licensed by the Nuclear Regulatory Commission (NRC), or have been approved by the host nation if the range is located overseas. Table 2-13 shows a list of current combat systems and their associated DU munitions.

Table 2-13. List of Depleted Uranium Munitions

Tank Ammunition 105 mm	**Tank Ammunition 120 mm**	**Bradley Fighting Vehicle 25 mm**	**A-10 30 mm**	**Harrier 25 mm**	**Phalanx 20 mm**
M774	M827	M1919	PGU-14/B	PGU-20	MK-149
M833	M829		PGU-14A/B		
M900	M829A1		PGU-14B/B		
	M829A2		PGU-14A/A		

c. The radiological hazards associated with DU munitions have been evaluated using M1919 25mm cartridge for the Bradley fighting vehicle and from practical experience during Operation Desert Storm. Since DU is mainly a very low alpha emitter, ingestion through inhalation or wounding is of the primary concern.

(1) Hazard classification testing was conducted on the M1919 25-mm APFSDS-T ammunition in 1988 at Nellis Air Force Base. Environmental sampling showed no indication that DU oxide had become airborne during a burn test where a pallet of ammunition was set on fire. Essentially all of the oxide produced was insoluble when analyzed using a simulated lung fluid test. Only 0.1 to 0.2 percent of the oxide was small enough to be inhaled. External radiation assessment was conducted in 1989. The components of the round itself and its packaging and shipping material effectively shielded out the predominant alpha and the beta radiation. However, gamma radiation penetrated both the components of the round and the shipping materials, with the highest radiation levels noted at the center of the shipping container. Radiation levels at the surface of a single shipping container had a maximum level of 0.6 mR/hour. This exceeds the safe surface exposure rate criteria of 0.5 mR/hour, but the shipment of this ammunition has been excepted from this criteria.

(2) When a kinetic energy penetrator strikes armor plating, a pyrophoric effect occurs. That is, a very fast moving, dense heavy metal penetrator striking steel armor will produce a white-hot ignition (flash) at the point of penetration. This pyrophoric effect occurs with either a conventional tungsten carbide penetrator (although to a lesser extent) or a DU penetrator. With a DU round, the penetration process generates high concentrations of airborne, breathable, DU oxides and high velocity shards of metal that can cause serious wounds. Data gathered from friendly fire incidents generally show that personnel must be in, on, or near (less than 50 meters) the target vehicle to internalize DU by inhalation or wound contamination. Almost as soon as the round hits and the dust has settled, the radiation levels on the outside of the vehicle will rapidly fall to levels that are much lower than the safety standards prescribed by the Occupational Safety Health Administration (OSHA) and the NRC. See Chapter 6, Section VII, for the treatment of personnel wounded by DU munitions. Personnel working with intact, unfired, DU rounds on a DU armored tank will not exceed, even if they slept in the vehicle, the occupational safety standards established by OSHA.

d. The radiological and toxicological risks of internalization of DU are currently under study. Internalization of DU through inhalation of particles in dust and smoke, ingestion of particles, or wound contamination may present potential radiological and toxicological risks. Battle damage assessment and recovery personnel who spend an extensive amount of time per year doing work in and on destroyed coalition and Iraqi vehicles should adopt protective measures. These include wearing gloves, rolling down sleeves and wearing an approved dust mask. These precautions are also recommended to prevent inhalation of all the other dust and debris that are in and on destroyed vehicles. Scientific data now demonstrate skeletal and renal deposition of uranium secondary to implanted DU fragments; however, there is uncertainty over the toxic level for long-term chronic exposure to internal uranium metal. It should be noted that no renal damage has been documented to date (see Chapter 6, Section VII).

2-15. Hazards of Nuclear Weapons Incidents

Nuclear weapons incidents can be both a peacetime and wartime problem. All elements of a military medical service should be prepared to provide the medical support as required if they occur.

a. Accidental Detonations.

(1) *High explosive detonations.* All nuclear weapons contain a conventional high explosive component. In any accident, there is a risk of either an explosion of this material or a fire. Either may occur immediately at the time of the accident, or later if a weapon is severely damaged and fragments of high explosive and nuclear material are scattered. All personnel at an accident site must be aware of these hazards and conduct all operations and duties under the direction of experienced ordinance disposal personnel. Nuclear detonations due to accidents and/or mishandling are considered to be highly unlikely.

(2) *Types of radiation.* The principle fissionable materials in nuclear weapons (^{235}U and ^{239}P) are basically alpha particle emitters. However, there are several weak (up to 185 keV) X and gamma ray emissions associated with alpha particle decay. The radiation intensity of X and gamma radiations at an accident site is generally low. The weak X and gamma radiations from unfissioned bomb material are not very penetrating, and the intensity is reduced by approximately 1/2 by 5.0 mm of tissue or water. The principal hazard is from airborne alpha particle emitters.

(3) *Potential injuries.* The victims of such accidents may have serious injuries, frequently multiple, requiring early, skilled treatment. These will include burns, fractures, head injuries, and so forth, typical of those sustained in serious accidents of all types. Significant radiation injury will not be present. Contamination of the injured with varying amounts of radioactive material may be present, but this contamination should not be a serious, immediate hazard to either the injured or to personnel caring for them. The number of accident victims may vary from very few, to a large number depending on the circumstances of the accident.

b. Explosive and Fire Hazards. As noted above, there is a significant risk of high explosive detonations and/or fire at a nuclear weapons accident site. This is increased in vehicular or aircraft accidents where oil or gasoline is present; and, as a result, burns would be a frequent and serious problem among the casualties of a nuclear weapons accident. If there is a fire, the smoke will contain a large variety of burned material from the weapons, the transport vehicle, and the environment. Some of these materials can be dangerous if inhaled. The particle sizes in the smoke from a fire will be important; a percentage of particles smaller than about 10 microns, if inhaled, may penetrate deeply into the respiratory system where the probability of retention is high, which can result in significant damage to the lungs.

c. Internal Hazards. At the typical accident site, there will be no significant external radiation hazard. A significant internal hazard can be present both early and late. The inhalation hazard is more serious immediately after an accident and during a fire, or following an explosion of conventional explosives when the plume may contain a percentage of respirable particles. Even though they are diluted by dispersion, these particles may be inhaled if they are resuspended as dusts into the atmosphere by winds or by the movement of people and vehicles. The concentration of particles per unit volume of air under these circumstances will be much less than that in the smoke of the burning weapon, and the particle size distribution will be different.

The actual substances which may be inhaled include a wide variety of both radioactive and nonradioactive materials.

d. Contamination of the Injured with Hazardous Material. Contamination of injured personnel may be due to either radioactive or toxic materials. In general, the hazard to both the patient and attending medical personnel will be so negligible that necessary medical or surgical treatment must not be delayed because of possible contamination. Decontamination should be done as soon as possible during the care of such patients, and ideally, prior to admission to a treatment facility. However, this will not always occur, and decontamination procedures should be part of the operational plans and orders of all divisions and departments of MTFs, not just of emergency services or first responder teams. This ensures flexibility of response and action and will prevent delay in needed medical treatment. The simple removal of outer clothing and shoes will, in most instances, affect a 90 to 95 percent reduction in the patient's contamination.

e. Contamination of Geographical Area Around Accident with Potential Hazard to Local Population. Medical personnel may be called upon to give advice as to the nature and degree of the public hazard associated with a given type and level of contamination. This hazard will rarely be an acute one but may well be a significant long-term one. The advice given will be an essential factor in determining what methods are used to minimize and remove the hazard.

(1) The most probable hazard will occur downwind from an accident site due to airborne particles of radioactive material which could be inhaled. Early after an accident, adequate information may not be available to determine the exact degree of the hazard from airborne contamination. As a result, a decision to evacuate an area close to an accident location may have to be made by local authorities without waiting for radiation measurements. No precise guidance can be given for these types of situations.

(2) A much less frequent hazard would be contamination of water supplies if an accident occurred near a river or reservoir. Dilution and settling of insoluble materials would further reduce this small hazard, and simple monitoring measures by trained personnel should be obtained before condemning the water supply. Water from other locations can be used temporarily until adequate measurements are made to determine whether there has been contamination or not. However, drinking water contaminated by plutonium is an insignificant hazard.

f. Contamination of Hospital Facilities, Equipment, and Personnel. Since the treatment of injured, contaminated personnel may result in the contamination of almost any part of an MTF, medical personnel must identify and measure the extent of contamination and then remove the contamination. The removal of contamination is a two-part problem and includes decontamination of personnel as well as decontamination of equipment and facilities. The former must be started as soon as possible, even if monitoring facilities are not available. Standardized procedures of decontaminating personnel must be established and instituted. Personnel must not be released before they have been monitored and decontaminated completely. The monitoring capability would be obtained from the technical teams working at the accident site. This requires coordination and communication with the authorities responsible for overall management of the nuclear accident.

2-16. Examples of Nuclear Incidents and Low-Level Radiation Emissions

It is extremely difficult to predict with any accuracy, the type and quantity of radionuclide contamination from a nuclear reactor accident, or an incident involving a dispersal of fissile material from a nuclear weapon or an RDD. However, one can look at previous incidents to get an estimate of the potential radionuclide contamination and the measures used to control the situation.

a. The Oak Ridge Plutonium Release of 1959. In November of 1959, a chemical explosion occurred during the decontamination of an evaporator in one of the shielded cells of a radio-chemical processing pilot plant at the Oak Ridge National Laboratory. The explosion is thought to have resulted from the formation of compounds, such as picric acid, when concentrated hot nitric acid was mixed with a decontaminating agent that contained phenol. The explosion resulted in plutonium contamination of the pilot plant building, nearby streets, and building surfaces. The adjacent air-cooled graphite reactor building became contaminated when plutonium was drawn into the ventilation system. Also, quantities of ^{95}Zr and ^{95}Nd were released.

b. The Palomares Incident. In January 1966, a B-52 bomber carrying four hydrogen bombs collided in midair with a KC-135 tanker near Palomares, Spain. The accident occurred during a routine high altitude air refueling operation as the B-52 was returning to Seymour Johnson Air Force Base in Goldsboro, North Carolina, after flying the southern route of the Strategic Air Command air alert mission code named "Chrome Dome." The bomber was attempting its third refueling try with a KC-135 tanker from the American base at Moron, when the nozzle of the tanker's boom struck the bomber. The boom ripped open the B-52 along its spine, snapping the bomber into pieces. The KC-135's 40,000 gallons of jet fuel ignited, killing all four tanker crew members and seven bomber crewmen; four of the bomber's crew parachuted to safety.

(1) Of the four H-bombs aboard, two of the weapons containing high explosive material exploded on ground impact, releasing radioactive materials, including plutonium, over the fields of Palomares (see Figure 2-4). A third nuclear weapon fell to earth but remained relatively intact; the last one fell into the ocean. Within hours, the DOD began to investigate the accident and began the operation to recover the weapons. It set up a recovery operation code named "Broken Arrow," the official designation for any operation involving a missing or damaged US nuclear weapon.

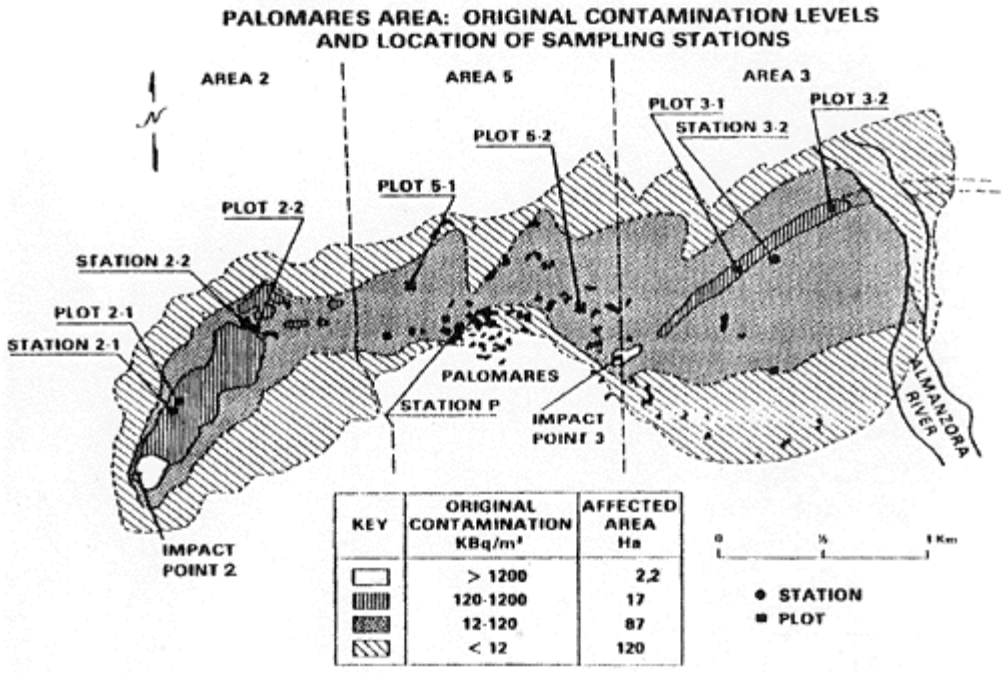

Figure 2-4. Palomares contamination levels.

(2) The weapon that sank in the Mediterranean set off one of the largest search and recovery operations in history. The search took about 80 days and employed 3,000 Navy personnel and 33 Navy vessels, not including ships, planes, and people used to move equipment to the site. Although the midget sub "Alvin" located the bomb after 2 weeks, it was not recovered until 7 April 1966. Wreckage from the accident fell across approximately 100 square miles of land and water. The most sophisticated computers of the time located at Sandia Laboratories calculated wind conditions, reverse trajectories, and so forth. However, it was not until Francisco Simo Orts, a local fisherman, provided critical information that the general location of the fourth bomb could be determined.

(3) The site was remediated within weeks of the accident. Where total soil surface contamination values exceeded 32 $\mu Ci/m^2$, contaminated vegetation and the surface layer (the top 10 centimeters [cm]) of soil were removed and shipped in metal drums to the Savannah River Site, South Carolina, and buried there (1,600 tons). Arable soil contaminated between 0.32 and 32 $\mu Ci/m^2$ (as determined by surface contamination values) was watered down and plowed to 30 cm deep. This served to dilute contaminated with uncontaminated soil to reduce surface contamination of radionuclides. On rocky hills in Impact Zone #2, where mechanical plowing was not possible, soil contaminated above 3.2 $\mu Ci/m^2$ was removed to the extent possible by hand tools. The exteriors of homes were hosed down with water to remove surface contamination.

(4) The Palomares incident remains today the most severe accident in US nuclear weapons history. Recovering bomb number four alone cost more than $50 million.

About 4,000 Air Force, Navy, and Army personnel and civilians participated in the recovery and reclamation. Ironically, the accident taught the US and nuclear weapons laboratories a lot about nuclear safety. Although the high-velocity ground impacts of two of the Palomares bombs resulted in the detonation of high explosives and the contamination of hundreds of acres of farmland with plutonium dust, weapons experts were reassured in the fact that neither the collision nor the ground impacts caused a nuclear detonation. Still, as a result of the accident, improvements were made to the safety of US weapons, including the continued development of insensitive high explosives.

c. Atomic Energy Commission Storage Igloo, Medina Base, San Antonio, Texas. On November 13, 1963, three employees were dismantling the high explosive components of a nuclear bomb; the components began burning spontaneously triggering a large blast involving 120 pounds of high explosives. The explosion caused little contamination. New York University's Dr. Joel Larus, who investigated the incident, was provided details of three similar incidents by the Atomic Energy Commission (AEC) on January 13, 1966. They are as follows:

- Hamburg, New York (January 4, 1958). An eastbound nickel plate railroad freight train derailed. Five cars carrying *AEC classified material* were involved in the accident. According to the report, there was no damage to the material and no injury to AEC personnel escorting the shipment.

- Winslow, Arizona (November 4, 1961). A trailer truck caught fire while carrying a small amount of radioactive material. There was no contamination resulting from the fire.

- Marietta, Georgia (December 2, 1962). A Louisville and Nashville train derailed while carrying nuclear weapons components. The material was not damaged, but three couriers were injured.

d. The Three Mile Island Incident. The Three Mile Island incident started on March 28, 1979 in Pennsylvania, and was due to a failure in an auxiliary component in the secondary system. The failure in this system led to loss of the water supply to the steam generators, which remove the heat produced by the nuclear reaction. An operator erroneously shut down the safety injection pumps and the coolant began to boil. Soon thereafter, the reactor coolant boiling uncovered the reactor core, and residual heat removal ceased. Part of the fuel melted, carrying fission products through the primary system into the pressurizer relief tank. This tank burst open under the rising pressure and fission gasses were released into the containment, actuating all of the radioactivity alarms. Radioactive water flowed down to the bottom of the reactor building, where it was to be collected for removal to an auxiliary building. After several confusing hours, the operator finally restored water injection to the reactor and reflooded the core. The reaction between the water and the heated cladding alloy led to the formation of hydrogen. The hydrogen gas mixed with air in the containment and caused an explosion with no serious consequences. Before the operator finally isolated the containment, fission gases, such as xenon and krypton, escaped through the ventilation filters. However, there was no uncontrolled release of iodines or other aerosols since they were all trapped in the water and the filters. Also, the accident had no biological effects in the surrounding population.

e. *The Chernobyl Incident*. On April 26, 1986 the Chernobyl power station located in the Ukraine, about 90 miles from Kiev, was destroyed in a catastrophic accident (see Appendix D, Health Effects and Casualty Treatment at Chernobyl). The accident occurred during the running of safety test, not during the normal operation of the reactor. The test carried out at Chernobyl-4 was designed to demonstrate that during an external electrical grid failure, a "coasting" turbine would provide sufficient electrical power to pump coolant through the reactor core while waiting for electricity from the back-up diesel generators. The circulation of coolant was expected to be sufficient to give the reactor an adequate safety margin.

(1) The scheduled lowering of the external grid power level began on schedule, and continued to be gradually lowered over a period of approximately 24 hours. At this time, the power level had decreased to 500 MW(t). It should be noted that the safe operating level for a pre-accident reactor of this type is about 700 MW (t). Control was transferred from the local grid to the automatic regulating system. Either the operator failed to give the "hold power at required level" signal, or the regulating system failed to respond to the signal. This resulted in a rapid drop in power to 30 MW(t). The operator then retracted a number of control rods to boost the power. Station safety procedures required that approval of the chief engineer be obtained to operate the reactor with fewer than the equivalent of 26 control rods. There were fewer than 26 after the operator retracted rods to get more power. Approximately 30 minutes later, reactor power had risen to 200 MW(t). Also as part of the test, two additional pumps were placed into operation to remove heat from the core more quickly. This reduced the water level in the steam separator, while the operator increased the water flow to maintain cooling. Thus began a cycle of repeatedly reducing the number of control rods to increase power, while reducing the feedwater flow to below normal in order to stabilize the steam separator level. This decreased heat removal from the core and resulted in a spontaneous generation of steam in the core.

(2) The actual test began when the turbine feed valves were closed to start turbine coasting. The automatic control rods were withdrawn from the core, but the expected decrease in steam quantity did not occur because of the reduced feedwater to the core. Approximately 30 seconds after the test began, steam in the core started to increase uncontrollably. The emergency button was pressed by the operator and control rods started to enter the core. The insertion of rods from the top concentrated all of the reactivity in the bottom of the core, allowing the reactor power to rise to 100 times the design parameters. Within seconds, fuel pellets shattered, reacting with the cooling water to produce a pulse of high pressure in the fuel channels, which ruptured 45 seconds after test initiation. Two explosions occurred--one was a steam explosion; the other was an explosion of the fuel vapor. The explosions lifted the nuclear pile cap, allowing the entry of air, which reacted with the graphite moderator blocks to form carbon monoxide. This gas ignited and a reactor fire resulted.

(3) The end result was that about eight out of the 140 tons of fuel, which contained plutonium and other highly radioactive materials (fission products), were ejected from the reactor along with a portion of the graphite moderator, which was also radioactive. These materials were scattered around the site, while cesium and iodine vapors were released by the explosion and fire. In summary, the factors that led to the accident were--

- Nonroutine operation of the reactor.
- Violation of operating regulations, including removal of most of the control rods.
- Positive void coefficient characteristic of the reactor.
- Apparent lack of knowledge of the station staff on the characteristics of the reactor.
- Inadequate control rod design.

f. *The Ukraine Rivne Nuclear Power Plant*. In January, 2000, a worker at Ukraine's Rivne nuclear power plant tried to switch off reactor number two by opening a safety valve in the cooling system. Duty engineers executed emergency measures that prevented the stoppage of the reactor. Rivne is 320 km (190 miles) west of the capital Kiev, and security was immediately tightened at the plant. Nervousness in the region remains high over the safety and security of Soviet-made reactors at Ukrainian plants since the Chernobyl incident in 1986. There were also several other security incidents in this region just before the Rivne incident. In December, 1999, a man tried to ram his car into the gates of a power station in southern Ukraine but guards shot and wounded him. Officials were also forced to shut down a reactor at the same plant later in December after a worker stole the cable which controlled the turbine.

CHAPTER 3

ENERGY PRODUCTION AND IONIZING RADIATION

Section I. MECHANISMS OF ENERGY PRODUCTION

3-1. General

As a first step in developing an understanding of the medical aspects of nuclear casualties and low-level radiation injuries, it is essential to understand how a nuclear weapon functions and how ionizing radiation is produced. Accordingly, a comparison will be made in this section between the mechanisms of energy production in conventional and nuclear detonations. In Section II, certain principles of ionizing radiation are presented to aid in the understanding of these concepts.

3-2. Conventional Chemical Explosives

The molecules of conventional chemical explosives are considered to be in a high-energy or unstable state. When such a system is made to react, products of greater stability are formed and energy is released. With a conventional explosive, such as TNT, the energy is derived from a sudden, violent chemical reaction, altering various bonds between the molecules of the explosive's chemical compounds. The amount of energy released in such a reaction is directly proportional to the difference between the total binding energy contained within the initial, unstable system and that contained within the final, more stable system. This net energy release is called the heat of explosion.

3-3. Nuclear Detonations

Energy released in a nuclear explosion is not produced by chemical reactions. Rather, it results from a nuclear reaction; either fission and fusion, in which fundamental changes occur in the composition of the nuclei of the reacting material rather than in the electron shells as is the case in chemical reactions. In these nuclear reactions mass is actually converted to energy and the amount of energy produced is many orders of magnitude greater than that available from chemical reactions. To fully appreciate the nature of these reactions, certain basic concepts related to atomic structure and nuclear reactions must first be understood.

3-4. Matter

Matter is the material substance in the universe that occupies space and has mass. All matter in the observable universe is made up of various combinations of separate and distinct substances called elements. An element is one of 106 known chemical substances, each of which cannot be broken down further without changing its chemical properties. Some examples are hydrogen,

nitrogen, silver, gold, uranium, and plutonium. The smallest particle of an element is called an atom. The structure of a helium atom is shown in Figure 3-1.

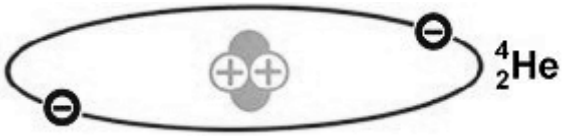

Figure 3-1. Helium atom.

a. Atomic Structure. The atom consists of a heavy, positively charged nucleus in which practically all of the mass of the atom is concentrated, and around which negatively-charged electrons revolve in closed orbits (see Figure 3-2). Atoms in their normal state are electrically neutral because the total negative charge of electrons outside the nucleus equals the total positive charge of the nucleus. The atomic nucleus is composed of two types of elementary particles called protons and neutrons, which are collectively known as nucleons. A proton is about 1,837 times heavier than an electron and possesses a positive charge equal in magnitude but opposite to the negative charge of the electron; a neutron is an uncharged particle and is about as massive as a proton. The number of protons in the nucleus is termed the atomic number of the element.

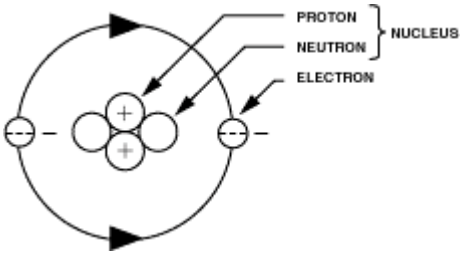

Figure 3-2. The structure of a typical atom.

b. Isotopes. Any grouping of nucleons capable of more than a fleeting existence is called a nuclide. Nuclides having equal numbers of protons but different numbers of neutrons are called isotopes (see Figure 3-3). The total number of nucleons in the nucleus is called the mass number of the nucleus. All nuclides with atomic numbers greater than 83 are unstable and spontaneously decay by radioactive disintegration. The radioactive disintegration process, called radioactivity, is the spontaneous emission of radiation from the nucleus of an unstable nuclide.

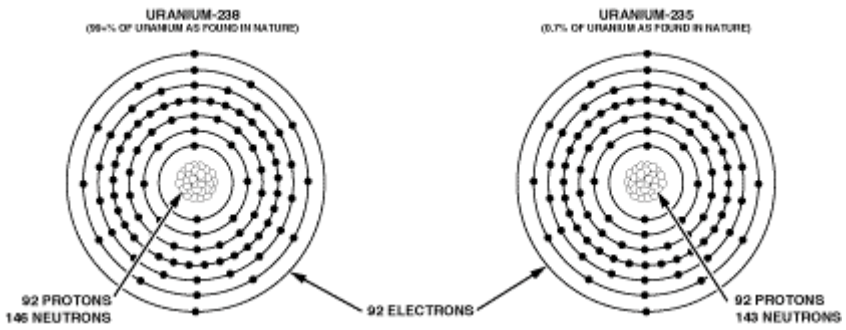

Figure 3-3. Two isotopes of uranium.

c. Symbols and Notation. A standard notational form is used to identify the individual isotopes of a given element. The standard notation takes the following form:

$$ {}^{A}_{Z}X $$

where X--chemical symbol of the element, Z--atomic number, and A--atomic mass number. An example of the standard for notation would be--

$$ {}^{235}_{92}U $$

Reference to a chart of the nuclides would reveal that the element with an atomic number of 92 is uranium, the chemical symbol for which is U. The atomic mass number 235 identifies a uranium isotope having 92 protons and 143 neutrons (235 -92=143) in its nucleus. Thus the isotope identified by the example notation is the naturally occurring, readily fissionable isotope of uranium used in nuclear weapons. The atomic number is frequently left off, and such an isotope may then be represented only by its mass number and chemical symbol, that is, ^{235}U.

3-5. Nuclear Reaction

In a nuclear explosion, nuclear potential energy is converted into the kinetic energy of the products of a process called nuclear fission. This process causes a release of energy that ultimately gives rise to the blast and thermal effects of a nuclear explosion, effects that are many orders of magnitude greater than those from a chemical explosion. In a nuclear power plant, the same fission reaction is used to produce electricity. But in the case of power production, the fission reaction is controlled so the energy is not released as an explosion.

a. Mass and Energy. Einstein's mass-energy equation, E--MC^2, shows that mass and energy are manifestations of a single entity and are interchangeable. The factor "C^2", the square of the speed of light in a vacuum, is the conversion factor relating units of mass and energy. This equation predicted the possibility of releasing enormous amounts of energy by the

conversion of mass to energy. Many experiments have demonstrated the universal validity of the equation.

b. *Mass Defect*. The nuclear potential energy released in the fission process originates from the binding energy of the nucleus. The binding energy comes from what is termed the "mass defect" of a nucleus. If the mass of all the nucleons (protons and neutrons) were measured, it would be more than the mass of the whole nucleus. So the mass defect is the difference between the mass of the nucleons and that of the nucleus. This is possible because some of the mass of the nucleons is converted into energy to hold them together. The amount of energy holding the nucleus together can be determined by Einstein's mass-energy equation where M is the mass defect.

3-6. Fission

Fission is a nuclear process in which a heavier unstable nucleus divides or splits into two or more lighter nuclei, with the release of substantial amounts of energy. The materials used to produce nuclear explosions by fission are those isotopes of uranium or plutonium which undergo fission most readily. These are ^{235}U and ^{239}Pu. When a free neutron of the proper energy is captured by the nucleus of a fissionable atom, the resulting unstable nucleus will *split* producing two or more fission fragments (atoms of different elements formed from the protons, neutrons, and electrons originally comprising the nucleus before its fission), two or three free neutrons and a tremendous amount of energy (see Figure 3-4).

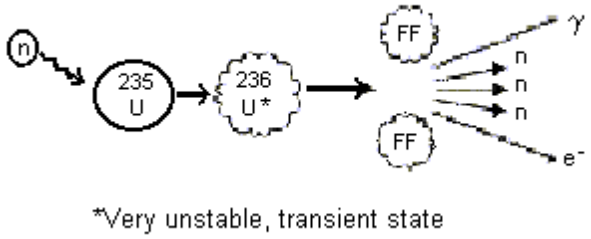

Figure 3-4. Fission of ^{235}U.

a. In terms of continued energy production, the most significant point about the fission process is the emission of free neutrons, which can in turn produce other fission events, which in turn produce still another generation of free neutrons. Each generation of fission-produced neutrons can produce a large number of fissions; and so, within a few generations, the total number of fissions produced can be tremendous. One of the easiest isotopes to fission is ^{235}U. When a thermal neutron is absorbed by a ^{235}U nucleus, it becomes ^{236}U. At this point, the ^{236}U either emits a gamma ray or undergoes fission. On the average, the ^{236}U nucleus will split into two smaller parts called fission fragments, release 2.5 neutrons, and emit gamma radiation (see Table 3-1).

Table 3-1. Fission Energy Partition

Average Energy Partition Fission of ^{235}U by Thermal Neutrons (total energy released per fission: 205 MeV)	
Radiation Type	%
Kinetic energy of fission fragment	82.0
Prompt gamma	2.5
Prompt neutron	3.5
Decay product: beta	4.0
Decay product: gamma	3.5
Decay product: neutron	5.0

b. While in principle, a single neutron could initiate a chain reaction of nuclear fissions that could ultimately result in the splitting of each fissionable atom in a given mass, not all of the neutrons produce more fissions. Some of the neutrons may escape from the fissionable mass while others may be removed by nonfission reactions. To initiate a chain reaction, sustain that reaction for a period sufficiently long to permit a buildup of explosive energy, and confine the released energy for as long as possible to maximize the weapon's explosive effect requires that a variety of special conditions be met.

c. Although fission events release more than two million times more energy per event than do chemical reactions, there still must be a tremendous number of fissions to result in the release of a significant amount of energy. To meet this requirement, a mass of fissionable material having specific characteristics must be assembled. Depending on size, and other factors, a given mass of fissionable material may support one of three types of chain reactions.

- Subcritical chain reaction. A reaction in which the number of neutrons decreases in succeeding generations, thus, the reaction does not continue.

- Critical chain reaction. A reaction in which the number of neutrons remains constant in succeeding generations.

- Supercritical chain reaction. A reaction in which the number of neutrons increases in succeeding generations.

3-7. Fusion

In general, fusion may be regarded as the opposite of fission. It is the combining of two light nuclei to form a heavier nucleus. For the fusion process to take place, two nuclei must be forced together by enough energy so that the strong, attractive, short-range, nuclear forces overcome the electrostatic forces of repulsion. The two conditions necessary for the fusion of appreciable numbers of nuclei are high temperatures to accelerate the nuclei, and high-pressure density to

increase the probability of interaction. The only practical way to obtain the temperatures and pressures required is by means of a fission explosion. Consequently, weapons with fusion components must contain a basic fission component (see Chapter 1). The energy released in the explosion of a fission-fusion weapon originates in approximately equal amounts from the fission and fusion processes.

Section II. IONIZING RADIATION

3-8. Nuclear Radiation

The nuclei of certain naturally occurring isotopes, and of others produced artificially, contain excess energy; that is, they are unstable. To attain stability, nuclei with excess energy emit that energy in the form of nuclear, ionizing radiation and, in that process, frequently change into different elements (see Figure 3-5). Isotopes, the nuclei of which emit ionizing radiation to achieve stability, are referred to as radioisotopes or radionuclides.

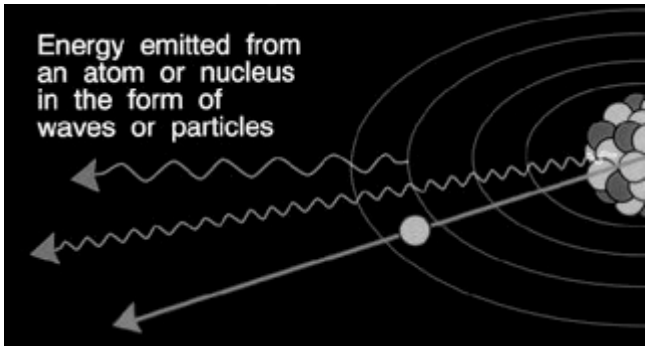

Figure 3-5. Radiation emissions.

a. Ionizing Radiation. Ionizing radiation is simply nuclear radiation in the form of particles or photons that carries enough energy to be able to strip electrons off of the atoms that the radiation interacts with. This leaves the atom with more protons than electrons, resulting in a free electron moving about and a net positive charge in the atom. This process is called ionization. The positively charged atom is referred to as a positive ion, and the negatively charged free electron is referred to as a negative ion. Ionization can be caused either by direct processes (for electrically charged radiation) or by indirect processes (for electrically neutral radiation).

b. Radioisotopes. Radioisotopes of heavy elements, such as radium or uranium, characteristically decay by emission of ionizing radiation in the form of alpha particles. Some heavy elements also decay by spontaneous fission which results in neutron releases. For the lighter elements, emission of beta particles is common. In addition, emissions of photons of

gamma radiation almost invariably accompany both alpha and beta particle radiation. Gamma or x-radiation (x-ray) constitutes the principal casualty-producing form of ionizing electromagnetic radiation associated with nuclear explosions. X-ray and gamma photons are essentially identical, differing only in their points of origin. Gamma photons originate in the nuclei of decaying atoms while x-rays originate in the electron shells surrounding nuclei.

(1) *Gamma rays and x-rays.* Even though they possess no net electrical charge, gamma and x-ray photons interact with atoms to produce ionization. They are pure energy and travel at the speed of light (3×10^8 meters per second). Gamma photons have discrete energies over a very wide range, but are considerably less ionizing than alpha or beta particles. However, gamma photons are much more penetrating. The primary mechanism for X-ray production is the bremsstrahlung process. The German word *bremsstrahlung* means *braking radiation*. When a beta particle passes close to the nucleus of an atom, it is deflected sharply from its original path by the strong coloumbic forces exerted on the beta particle by the nucleus. This change in direction causes the beta particle to decelerate, which in turn causes an X-ray photon to be emitted from the beta particle (Figure 3-6).

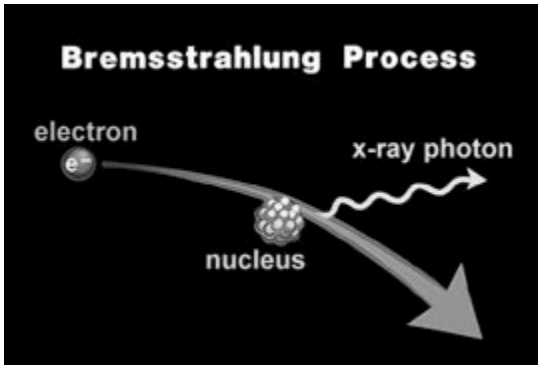

Figure 3-6. X-ray production.

Gamma rays, which originate in the nucleus of an atom, are emitted from an excited nucleus following an alpha or beta decay. For example, the emission of gamma rays from cobalt-60 actually follows a beta decay (see Figure 3-7).

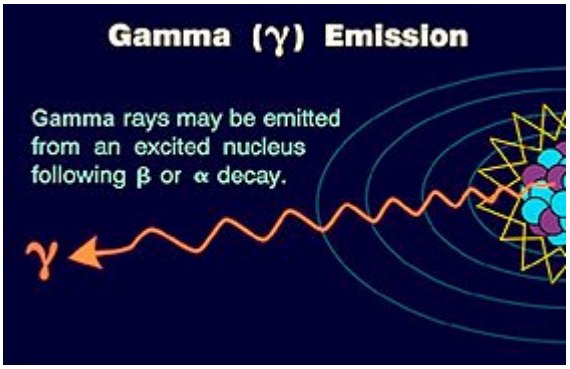

Figure 3-7. Gamma ray procuction.

(2) *Alpha particles.* An alpha particle is a completely ionized helium nucleus consisting of two protons and two neutrons all strongly bound together by nuclear forces (see Figure 3-8). Alpha particles are emitted by unstable atomic nuclei, have a mass about 8000 times that of electrons, and carry a strong, double-positive charge. Although highly ionizing, alpha particles are only slightly penetrating.

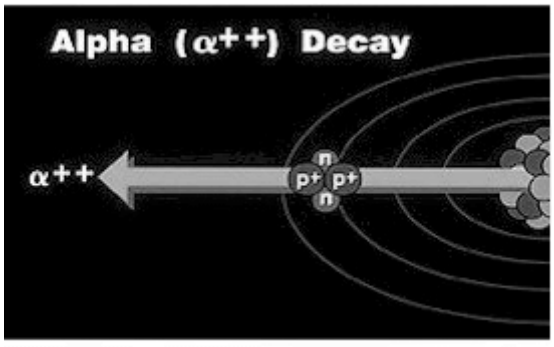

Figure 3-8. Alpha particle.

(3) *Beta particles*. Beta particles are identical to atomic electrons but, like alpha particles, they are ejected from a nucleus when the nucleus rearranges itself into a more stable configuration (see Figure 3-9). Beta particles interact with matter through collisions with atomic electrons. They can also be deflected at sharp angles by an atomic nucleus, causing a photon to be emitted via the bremsstrahlung process. As opposed to alpha particles, beta particles show continuous energy spectra and because of its smaller mass and charge, a beta particle is less ionizing than an alpha particle but relatively more penetrating.

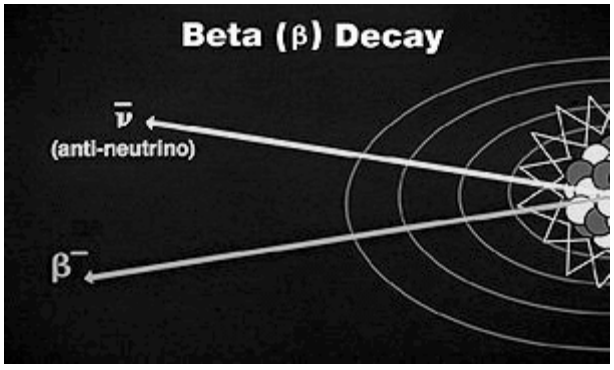

Figure 3-9. Beta particle.

(4) *Neutrons.* In the fission process, neutrons are also released and consequently, make up a significant portion of the total radiation output. Neutrons are

electrically neutral and interact with matter by either collisions with, or absorption by an atomic nucleus. Collisions with atomic nuclei slow down, or thermalize, a neutron so it may undergo nuclear capture. In nuclear capture, the incident neutron is actually absorbed into the nucleus. This can make the nucleus unstable and, therefore, radioactive (see Figure 3-10).

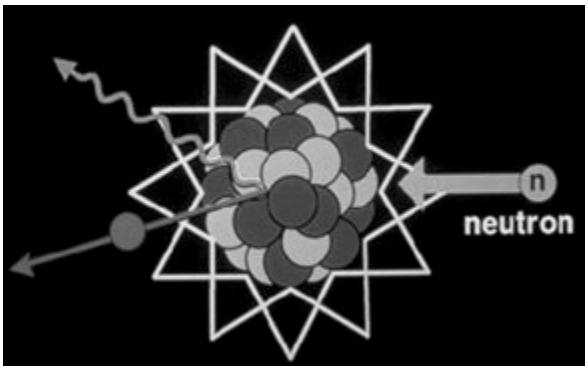

Figure 3-10. Nuclear capture of a neutron.

c. Radioactive Decay. The process wherein radionuclides emit ionizing radiation is also termed radioactive decay. Each radioisotope has its own characteristic decay scheme. A decay scheme identifies the type or types ionizing radiation emitted; the range of energies of the radiation emitted; and the decaying radioisotope's half-life. Depending upon the type of particulate radiation emitted, decaying nuclei can, in addition to changing their energy states, be transformed into new elements. Examples of the transformations resulting from alpha and beta particle decay are shown in Table 3-2.

Table 3-2. Radioactive Decay.

	Isotope	Half-Life	Radiation Emitted	Decay Products	Half-Life
Fissionable Material	Uranium-235 Uranium-238 Plutonium-239	7.1×10^8 yr 4.5×10^9 yr 2.4×10^4 yr	α , γ α , γ α , γ	Thorium-231* Thorium-234* Uranium-235*	25.2 hr 24 days 7.1×10^8 yr
Fission Products	Lanthanum – 140 Iodine-131 Strontium-90 Cesium-137	40 hrs 7 days 28.9 yr 30.0 yr	β^- , γ β^- , γ β^- , β^- , γ	Cerium-140 Xenon-131m Yttrium-90 Barium-137m	Stable 11.9 days 64 hr 2.5 min
Other Radioisotopes	Radon-222 Potassium-40	3.8 days 1.3×10^9	α , γ β^- , β^+ , γ	Polonium-218* Cesium-40	3 min Stable

		days		or Argon-40	Stable
	Sodium-24		β^- , γ	Magnesium-24	Stable
	Hydrogen-3 (Tritium)	1.5 hr 12.3 yr	β^-	Helium-3	Stable
* Includes other daughter radionuclides					

3-9. Units of Measure

There are several different, but interrelated, methods of measuring and quantifying ionizing radiation. The scientific community is now using the standardized International System of Units (*systeme international d'unites*, abbreviated internationally as SI), which is a version of the metric system. For comparison purposes, old and new units of measurement are discussed below.

a. Activity. The activity of a radionuclide is simply a measure of how many atoms undergo radioactive decay per a unit of time. The old unit for this is the curie (Ci), in honor of Marie Curie who discovered radioactivity while working with radium-226. The curie is based on the activity of 1 gram of radium-226, or 3.7×10^{10} radioactive disintegrations per second. The SI unit for measuring the rate of nuclear transformations is the becquerel (Bq). The becquerel is defined as one radioactive disintegration per second (see Figure 3-11). It is important to note that activity is not a measure of the quantity of a substance but of the rate of radioactive transformations occurring in that substance. Thorium-232 radioactively decays at a much slower rate than radium-226. Therefore, it would take a large amount of thorium-232 to have the same amount of activity as a small amount of radium-226.

Curie (Ci)
1 Ci--3.7×10^{10} nuclear transformations per second

Becquerel (Bq)
1 Bq--1 nuclear transformation per second
1 Bq--2.7×10^{-11} Ci

Figure 3-11. Units of activity.

b. Exposure. The old unit for exposure is the roentgen (R). The roentgen is a measure of the number of ion pairs produced by gamma rays in a unit volume of air. Some radiation passes through a volume of medium without interacting and, therefore, does no damage. Thus, only the radiation that interacts with the medium is measured. The SI unit for exposure is the coulomb per kilogram, or unit of charge generated per unit mass.

c. Absorbed Dose. To look at biological effects, a measurement of how much energy is deposited per unit mass of the medium with which the radiation is interacting is

required. This quantity is known as the absorbed dose. The old unit of measure for this is the rad, which stands for *radiation absorbed dose*. The rad is defined as 100 electroretinograms (ergs) of energy deposited per one gram of medium. The SI unit of measure for absorbed dose is the Gy, which is defined as one joule of energy deposited per one kilogram of medium. Fortunately, it is easy to convert the two since 1 Gy equals 100 rads (see Figure 3-12).

Rad
1 rad = 100 ergs/gram
Gray (Gy)
1 Gy = 1 joule/kilogram = 100 rads

Figure 3-12. Units of absorbed dose.

d. Linear Energy Transfer. Not all ionizing radiation is equally damaging. How damaging any given radiation is depends on many factors. Two of the most important factors are (1) how much energy is deposited and, (2) how quickly the energy is deposited. To understand the factors involved in measuring radiation damage, the concept of linear energy transfer (LET) must be discussed. High LET radiation deposits all of its energy in a comparatively short distance so that each cell that is hit receives a higher portion of the total energy deposited than would be received from low LET radiation. Conversely, low LET radiation deposits its energy over a longer distance so that more cells are hit but each receives a lower portion of the total energy deposited (see Figure 3-13). To a considerable extent, the relative biological effectiveness (RBE) of various radiations depends on the rate of energy loss (LET), along the paths of the individual ionizing particles or photons. Radiations with low LET, such as x-rays or gamma rays, produce diffuse ionizations throughout the medium. In contrast, the LET associated with neutrons or alpha particles is so high that the passage of a single track will, in all probability, put enough ionizations into a traversed cell to produce death.

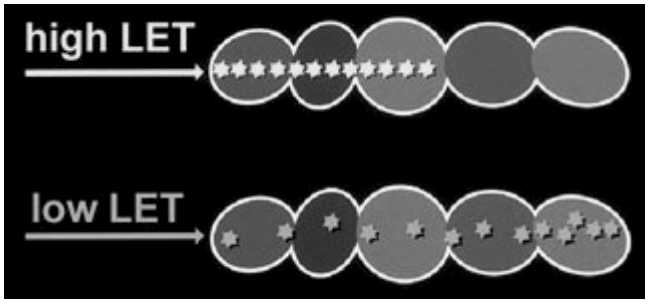

Figure 3-13. High LET versus low LET.

e. Weighting Factors. To account for differences in LET when measuring the effect of radiation, each type of radiation has been assigned a radiation weighting factor (W_R). This

was done by measuring how much of each radiation type it took to produce the same biological effect as 200-kilo electron volt (KeV) x-rays. As shown in Table 3-3, all photons, beta particles, and electrons do the same amount of damage. Thermal neutrons do somewhat more damage, and fast neutrons and alpha particles are extremely damaging. Indeed, of the common radiation types, alpha particles and high-energy neutrons are the most damaging.

Table 3-3. Radiation Weighting Factors

Radiation Type	**W_R**
X-ray	1
Gamma ray	1
Beta particle and electron	1
Neutron <10 KeV	5
Neutron 10 KeV to 20 MeV	10-20
Neutron >20 MeV	5
Alpha particle	20
Radiation weighting factors account for linear energy transfer (LET) differences.	

MeV--megaelectron volt.

f. Equivalent Dose. The Wr is used to determine the equivalent dose. The equivalent dose is a measure of the relative effectiveness of a given amount of absorbed energy from a particular type of radiation in causing a certain biological effect. The old unit of equivalent dose is the rem, which is equal to the absorbed dose, or rad, multiplied by the Wr. The SI unit is the Sv (see Figure 3-14). Note that the Wr is just a multiplicative constant. One rem is 100 ergs per gram, and 1 Sv is 1 joule per kilogram. Also, just as 1 Gy is 100 rads, 1 Sv is 100 rems. Hence, the Wr takes into account that some types of radiation are much more damaging than others.

Rem--W_R x Rad

Sv: 1 Sv--1 joule/kilogram--100 Rem

Figure 3-14. Units of equivalent dose.

3-10. Shielding

Personnel can be shielded from ionizing radiation by various materials. The most effective shield depends on the type and energy of the radiation (see Figure 3-15). Alpha particles, due to their strong interactions with atomic electrons, can be stopped by a piece of paper. On the other hand, a thicker or denser material, such as aluminum, is needed to stop beta particles.

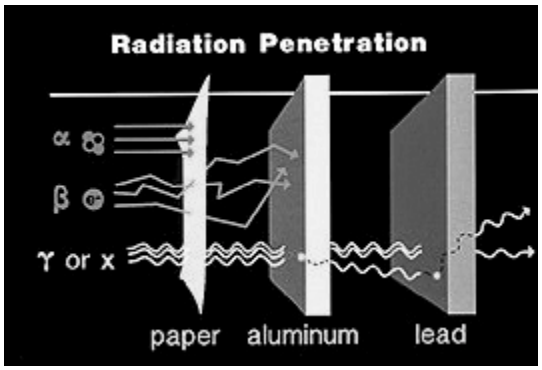

Figure 3-15. Radiation shielding.

a. Gamma rays and x-rays are even more difficult to shield against. A material with a large number of protons per atom (and thus a large number of electrons) is needed to limit the number of gamma and x-rays that penetrate the shield. Such materials are called *high Z* materials and a good example is lead. No matter how thick or dense a gamma or x-ray shield is, some of the photons will still get through.

b. Neutron shielding is a more complicated task due to the process of nuclear capture. To be effective, the shielding material must be able to slow down the neutrons. The best elements for this are *low-Z* elements such as hydrogen. After the neutrons are thermalized, there must be neutron-absorbing material present. The best example of this is boron. It is important to note that lead and other high-Z materials do not provide effective shielding against neutrons. In order for a neutron to be slowed down effectively, it must collide with something of similar mass. After the neutrons are gone, the radiation from the atoms that absorbed the neutrons must be shielded. For this, additional layers of the materials effective against alpha and beta particles, x-rays, and gamma rays must be used.

3-11. Interaction with Matter

Ionizing radiation interacts with matter in one of two ways--it is either scattered or absorbed. Both result in deposition of energy in the target system. The energy that is deposited by these reactions is of particular interest because it may result in physiological injury.

a. Transfer of Energy. Transfer of energy from an incident photon or particle to the atoms of an absorbing target material may occur via by excitation or ionization. Excitation involves the addition of energy to an atomic or molecular system, thereby transferring it from its

ground or stable state to an excited or unstable state. Depending upon the type of interaction, either the atomic nucleus or one of its orbital electrons may absorb the excitation energy. Ionization is the process which results in the removal of an electron (negative charge) from an atom or molecule thereby leaving the atom or molecule with a net positive charge. Ionization occurs if alpha or beta particles, or gamma photons transfer sufficient energy to dislodge one of the electrons from the outer orbital shells of the target atom. Each ionization event produces an ion pair consisting of a free electron and the positively charged remainder of the atom.

b. Gamma Interaction. In terms of ionization, gamma radiation interacts with matter via the processes of the photoelectric effect, Compton scattering, and pair production. These processes are not discussed in detail here. For all of these processes the end result is to cause the formation of a positively charged ion via the ejection of electrons.

c. Interaction of Charged Particles.

(1) Due to the high probability of interaction between an alpha particle and orbital electrons in an absorbing medium, a large number of ion pairs are formed per unit path length. Since a finite fraction of the total kinetic energy of an alpha particle is absorbed with the formation of each ion pair, the alpha particle will lose its energy over a relatively short distance. For these reasons, the range of alpha particles is much less than the range of beta particles. Table 3-4 illustrates this for a 37 kBq (1.0 Ci) source of an alpha emitter of moderate energy.

Table 3-4. Tissue Dose Rate at Various Distances Around a 37 kBq (1μCi) Alpha Emitter

Distance (μm)	Dose Rate at Distance (Gy/hr)
10	1.7×10^6
20	5.2×10^5
30	0

(2) Beta particles and orbital electrons have negative charges, resulting in electrostatic repulsion when in the vicinity of another like particle. Normally a beta particle loses its energy in a large number of ionization and excitation events in a manner analogous to the alpha particle. However, the range of the beta particle is considerably greater than that of an alpha particle. The beta particle travels longer distances between interactions and follows a torturous path through matter. Table 3-5 illustrates this for 37 kBq (1.0 Ci) sources of common beta emitters.

Table 3-5. Tissue Dose Rate at Various Distances Around

a 37 kBq (1.0 Ci) Particle of Various Beta Emitting Materials*

Distance	Carbon 14	Strontium 90	Phosphorus 32
10μm	2,000,000	766,400	380,000
100μm - 0.1 mm	1,500	7,380	3,700
200 μm - 0.2 mm	40	1,705	930
400μm - 0.4 mm	0.03	340	230
600μm - 0.6 mm	0	130	100
1,000μm – 1 mm	0	34	30
10,000 μm – 10 mm	0	0.02	0
Max Beta Energy (MeV)	*0.156 MeV*	*0.546-2.267 MeV*	*1.71 MeV*

*1 Bq--one disintegration per second (1 Ci--3.7×10^{10} Bq)
Dose rate in cGy/hr: Range in tissue 1-10 mm.

d. Specific Ionization. The penetrating ability of radiation depends on the rate at which the radiation deposits energy along its path. The term *specific ionization*, which is defined as the average number of ion pairs generated per unit length of path, is used to describe the ionizing capability of ionizing radiations. Generally speaking, the ion density along the path of a low-energy particle is greater than that along the path of a high-energy particle of the same mass and charge. This is because the low-energy particle is moving slower and has more time to interact. The ion density towards the end of the path of a particle is greater than at the beginning, because its velocity is less and the probability of interaction is increased accordingly. Alpha particles are capable of producing the highest specific ionization followed by beta particles and the secondary electrons produced by gamma-photon interactions (see Table 3-6).

Table 3-6. Specific Ionization of Radiation

Radiation	Range in Air	Speeds	Specific Ionization
Alpha (α)	5-7 cm	3,200 – 32,000 km/sec	20,000 – 50,000 ion pairs / cm
Beta (β)	200- 800 cm	25- 99% of the speed of light	50 - 500 ion pairs / cm
Gamma (γ)	Use of half-thickness(High-Z material)	Speed of light 300,000 km / sec	5 – 8 ion pairs / cm
Neutron (N)	Use of half-thickness (Low-Z		

	material)		

e. Stopping Power. The maximum ranges of ionizing radiation in matter depends not only on the characteristics of the specific particles but also on the stopping power of the absorbing material. The stopping power of a material is a function of electron density or the number of electrons per unit volume of the substance and represents the total energy lost in collision and radioactive interaction. Materials differ in their stopping power on the basis of the ratio of their atomic number to their atomic mass (Z/A) times the density of the material. The range of a charged particle in an absorbing material is inversely proportional to this ratio. For example, if the range of a given energy beta particle is 1 cm in water, its range in air would be much greater (about 10 meters), and in iron much smaller (about 1 mm).

f. Half-Value Layer. The concept of stopping power is not generally used in connection with material interactions of either gamma photons or uncharged neutrons. Since high-energy radiation is in general more penetrating than low-energy radiation, the specification of half-value layer (HVL) is often a convenient method of characterizing the penetrating quality of an energy spectrum. Half-value layer is defined as that absorber thickness which reduces a given radiation intensity to one-half of the incident value (see Figure 3-16). The relatively high penetrating power of X and gamma radiation compared with that of charged particles is related to the fact that the absorption interactions are fairly rare occurrences.

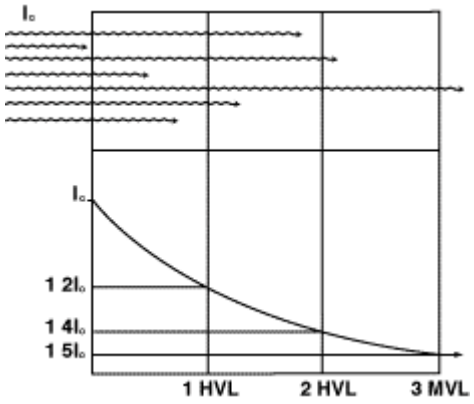

Figure 3-16. Attenuation of gamma radiation.

g. Neutron Interaction. Although most ionizing radiation injuries associated with nuclear warfare (NW) will be attributable to gamma radiation, a sufficient number of high-energy fission neutrons escape from the detonation to represent a significant hazard at considerable ranges. The neutron is a particle, and thus is fundamentally different from electromagnetic radiation. It also differs from other particulate radiations (alpha and beta) in that neutrons do not carry any electrical charge. As a result, neutrons do not interact with the orbital electrons of atoms, but instead interact directly with the nuclei of atoms, particularly those having low atomic mass numbers. Depending on their point of origin, neutrons may have

energies ranging from a fraction of an electron volt (eV) for so-called thermal neutrons to several megaelectron volts (MeV) for fission neutrons, to fusion neutrons which have energies of up to 14 MeV (for example, a deuterium-tritium reaction). Most neutrons produced in a nuclear fission detonation will have energies less than 1 MeV. A small fraction will have energies above 3 MeV. In enhanced radiation weapons, there will be a preponderance of 14 MeV neutrons.

h. Neutron-to-Gamma Ratios. The total dose due to initial radiation from a nuclear weapon can be divided into two components, neutrons and gamma rays. The neutron-to-gamma ratio is the ratio of neutron dose to gamma dose present at a specified point. The neutron-to-gamma ratio (n/γ) for a given total dose level (n+γ) is dependent on weapon yield and design, air density, and height-of-burst (HOB). Some typical neutron-to-gamma ratio values for 26 Gy total dose to an unprotected individual are shown in Table 3-7. As a general rule, the neutron-to-gamma ratio decreases with the range from the weapon's ground zero. This is due to the neutrons interacting with the air, creating secondary gamma radiation. As a result, the gamma component decreases at a slower rate than does the neutron component. These dose levels are typical of safety criteria. The ratios for vehicles and shelters depend on the specific neutron and gamma protection factors associated with the vehicle or shelter and are based on the material used in construction. There are no typical ratios for vehicles, since each component of the ratio is effected differently by the associated radiation protection factor. However, for a tank, the protection factors are about 2 and 10 for neutrons and gamma rays, respectively. In other words, the neutron component would be decreased by a factor of two. Therefore, at least for tanks, the gamma radiation is more effectively stopped. This will significantly affect the neutron-to-gamma ratio within the vehicle.

Table 3-7. Typical Neutron-to-Gamma and Neutron Dose-to-Total Dose Ratios

Yield (kT)	N/γ	n/n + γ	Range (Meters)
0.1	4.6	0.82	360
1.0	3.0	0.75	650
10.0	1.6	0.62	1040
100	0.47	0.32	1500
1000	0.042	0.04	2280
Assumptions: HOB--$60W^{1/3}$ meters, where HOB--height of burst; W--yield in KT; air density is equal to 0.9 (relative to sea level); fission-only device; total dose is equal to 2.6 Gy.			

CHAPTER 4

EFFECTS OF NUCLEAR EXPLOSIONS

Section I. GENERAL

4-1. Overview

The basic differences in the mechanism of energy production and related characteristics of conventional and nuclear detonations are discussed in Chapter 3. This chapter discusses how the energy produced by nuclear weapons affects the surrounding environment and lays the foundation for a discussion of the biological effects of blast and thermal radiation in Chapter 5. The most obvious difference between the detonation of a conventional weapon and the detonation of a nuclear weapon is the yield-to-weight ratio. The complete fission of one pound of uranium or plutonium releases as much explosive energy as does the explosion of 8,000 tons of TNT. Also, the location of the detonation is just as important as the weapon's yield in determining the energy distribution.

4-2. Nuclear Detonation

A nuclear detonation results from the formation of a supercritical mass of fissionable material, with a near instantaneous release of nuclear binding energies and large-scale conversion of mass to energy.

a. Basic Detonation Characteristics. The destructive action of conventional explosions is almost entirely due to the transmission of energy in the form of a blast wave and the resultant projectiles (shrapnel). The energy of a nuclear explosion is transferred to the environment in three distinct forms--blast, thermal radiation, and nuclear radiation. The energy distribution among these three forms will depend on the weapon yield, the location of the burst, and the characteristics of the environment. The energy from a low altitude atmospheric (endoatmospheric) detonation of a moderate-sized weapon in the KT range is distributed roughly as follows (see Figure 4-1):

- Fifty percent as blast.

- Thirty-five percent as thermal radiation, which is made up of a wide range of the electromagnetic spectrum including infrared, visible, and ultraviolet light and some soft x-rays.

- Fifteen percent as ionizing radiation, including 5 percent as initial (or prompt) radiation emitted within the first minute after detonation, consisting chiefly of neutrons and gamma rays, and 10 percent as residual nuclear radiation (fallout).

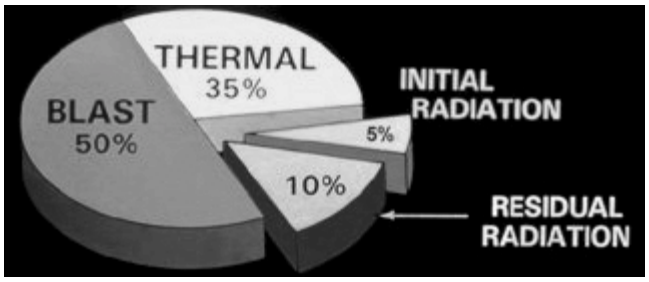

Figure 4-1. Energy partition from a nuclear detonation.

b. Range of Damage. Table 4-1 demonstrates the ranges of damage for weapons of various yields. The distribution of energy is significantly altered in the enhanced radiation nuclear weapon (neutron bomb). An enhanced radiation warhead is designed specifically to reduce the energy that is dissipated as blast and heat with a consequent increase in the amount of initial radiation. Its approximate energy distribution is 30 percent blast, 20 percent thermal, 45 percent initial radiation, and 5 percent residual radiation.

Table 4-1. Comparison of Weapons Effects Nuclear Effects Radii (Meters)*

Effect	1 kT	10 kT	100 kT	1000 kT (1MT)
Ionizing radiation (50% immediate transient ineffectiveness)	**600**	**950**	**1400**	**2900**
Ionizing radiation (50% untreated lethality)	**800**	**1100**	**1600**	**3200**
Blast (50% casualties)	**140**	**360**	**860**	**3100**
Thermal radiation (50% casualties, second degree burns under fatigue uniform)	**369**	**1100**	**3190**	**8020**

* Height of burst idealized for maximal blast effect.

4-3. Initial Energy Transfer and Formation of the Fireball

Because of the tremendous amounts of energy liberated per unit mass in a nuclear detonation, temperatures of several tens of millions degrees centigrade develop in the immediate area of the detonation. This is in marked contrast to the few thousand degrees of a conventional explosion. At these very high temperatures the remaining parts of the nuclear weapon are vaporized. The

fissioned atoms release most of their energy as kinetic energy of the fission products. By a series of local collisions, this energy is transformed into large amounts of electromagnetic radiation, consisting chiefly of soft X-rays. In an endoatmospheric detonation, this radiation is absorbed within a few meters of the point of detonation by the surrounding atmosphere. The atmosphere is heated to extremely high temperatures and forms a brilliantly hot sphere of air and gaseous weapon residues, the so-called fireball.

a. The fireball immediately begins to grow rapidly and rise like a hot air balloon. Within a millisecond after detonation, the diameter of the fireball from a one MT airburst is 150 meters. This diameter increases to a maximum of 2,200 meters within 10 seconds. The fireball is simultaneously rising at the rate of 100 meters/second. The initial rapid expansion of the fireball severely compresses the surrounding atmosphere, producing a powerful blast wave.

b. The fireball emits enormous amounts of electromagnetic radiation, similar in its spectrum to sunlight. This is usually termed thermal radiation. The visible light component accounts for the blinding flash seen upon detonation as well as the subsequent brightness of the fireball. The infrared component causes widespread burns and incendiary effects.

c. As it expands toward its maximum diameter, the fireball cools. After about a minute its temperature has decreased to an extent that it no longer emits significant amounts of thermal radiation. The combination of upward movement and the cooling of the fireball give rise to the formation of the characteristic mushroom-shaped cloud. As the fireball cools, the vaporized materials in it condense to form a cloud of solid particles. Following an airburst, condensed droplets of water give it a typical white cloud-like appearance. In the case of a surface burst, this cloud will also contain large quantities of dirt and other debris which are vaporized when the fireball touches the earth's surface or are sucked up by the strong updrafts afterwards, giving the cloud a dirty brown appearance. The dirt and debris become contaminated with the radioisotopes generated by the explosion or activated by the neutron radiation and fall back to earth as fallout.

d. The cloud rises for a period of approximately 10 minutes to a stabilized height that depends on the thermal output of the weapon and atmospheric conditions. It will continue to grow laterally, assuming the familiar mushroom shape and may remain visible for an hour or more under favorable conditions. For example, the nuclear cloud from a one MT surface burst will stabilize at an altitude of 20 km and will have a mean lateral diameter of 35 km.

4-4. Types of Bursts

The altitude at which the weapon is detonated will largely determine the relative effects of blast, heat, and nuclear radiation. Nuclear explosions are generally classified as airbursts, surface bursts, subsurface bursts, or high altitude bursts.

a. Airburst. An airburst is an explosion in which a weapon is detonated in air at an altitude below 30 km but at sufficient height that the fireball does not contact the surface of the earth. After such a burst, blast may cause considerable damage and injury. The altitude of an

airburst can be varied to obtain maximum blast effects, maximum thermal effects, desired radiation effects, or a balanced combination of these effects. Burns to exposed skin may be produced over many square km and eye injuries over a still larger area. Initial nuclear radiation will be a significant hazard with smaller weapons, but the fallout hazard can be ignored, as there is essentially no fallout from an airburst. The fission products are generally dispersed over a very large area unless there is local rainfall which would result in a more localized fallout pattern. In the vicinity of ground zero, there may be a small area of neutron-induced activity that could be hazardous to troops required to pass through the area. In the tactical nuclear arena, a discrete *package* of airbursts is most likely to be used against ground tactical formations.

b. Surface Burst. A surface burst is an explosion in which a weapon is detonated on, or slightly above, the surface of the earth so that the fireball actually touches the land or water surface. Under these conditions, the area affected by the blast, thermal radiation, and initial nuclear radiation will be less extensive than for an airburst of similar yield, except in the region of ground zero where destruction is concentrated. In contrast with airbursts, local fallout can be a hazard over a much larger downwind area than that affected by blast and thermal radiation. Tactical nuclear weapons would be employed in this manner in order to create large "hotspots" as obstacles to enemy unit maneuver.

c. Subsurface Burst. A subsurface burst is an explosion in which the point of the detonation is beneath the surface of the land or water. Cratering will generally result from an underground burst, just as for a surface burst. If the burst does not penetrate the surface, the only other hazard will be from ground or water shock. If the burst is shallow enough to penetrate the surface, blast, thermal, and initial nuclear radiation effects will be present, but will be less than for a surface burst of comparable yield. Local fallout will be very heavy if surface penetration occurs.

d. High Altitude Burst. A high altitude burst is one in which the weapon is exploded at such an altitude (above 30 km) that initial soft X-rays generated by the detonation dissipate energy as heat in a much larger volume of air molecules. The fireball is much larger and expands much more rapidly. The ionizing radiation from the high altitude burst can travel for hundreds of miles before being absorbed. Significant ionization of the upper atmosphere (ionosphere) can occur, with the intent of causing severe disruption in communications. High altitude bursts generate an intense electromagnetic pulse (EMP) which can significantly degrade performance of, or destroy sophisticated electronic equipment. For example, a high altitude burst of strategic weapons could be used to disrupt or destroy national command, control, communications, computers, and intelligence systems. There are no known biological effects of EMP; however, indirect effects may result from failure of critical medical equipment.

Section II. BLAST

4-5. Formation and Propagation of Blast Wave

The very high temperatures and pressures at the point of detonation cause residues to move outward radially from the center of the explosion with very high velocities. This transfers energy to the atmosphere by impulse and generates a steep-fronted, spherically expanding shock wave. A shock wave in air is generally referred to as a *blast wave* because it is accompanied by a strong wind. In water or in land, the shock is much like that of a sudden impact. The blast or shock wave behaves much like a moving wall of compressed air and is responsible for most of the material damage caused by a nuclear explosion. Objects within the path of the blast wave are subjected to severe, sharp increases in atmospheric pressure and to extraordinarily severe transient winds. Most buildings, with the exception of reinforced or blast-resistant structures, will suffer moderate to severe damage. The velocity of the accompanying blast wind may exceed several hundred km per hour (see Figure 4-2 for an example based on a one MT airburst).

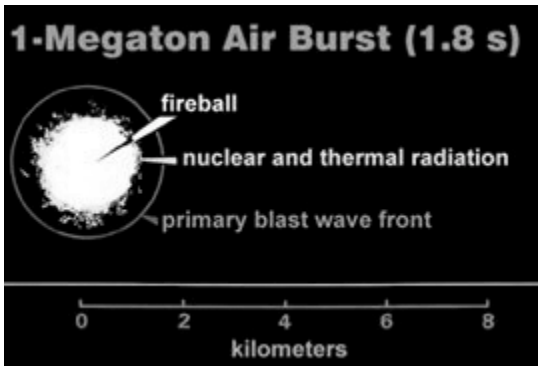

Figure 4-2. Production of a blast wave.

a. Primary Blast Wave. The primary blast wave continues to travel away from the point of explosion. At the point where the blast wave contacts the earth's surface, wind velocities reach or exceed 400 mph. When the lower portion of the blast wave contacts the surface, it is reflected in much the same way that an echo is produced when a sound wave strikes a surface. The reflected wave is also capable of producing material damage (see Figure 4-3). Also, ground shock waves will also be induced as a result of an airburst. For a very large overpressure in the blast wave, the ground shock will penetrate some distance into the ground and may damage underground structures, buried utilities, and so forth.

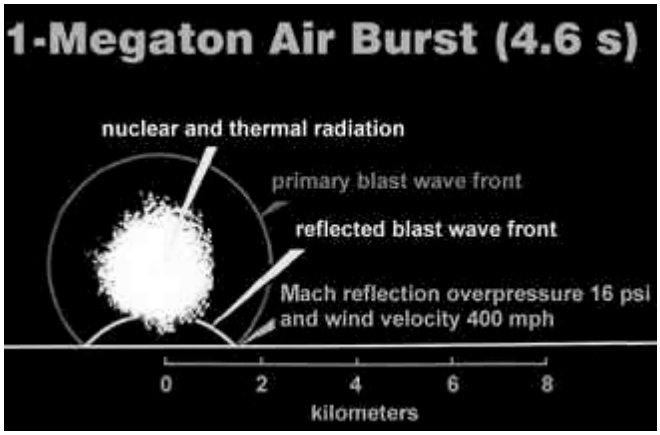

Figure 4-3. Blast wave reflection.

b. Mach Effect. At some point on the earth's surface, depending largely on the size of the blast, the reflected wave and primary wave fronts merge. This merging phenomenon is known as the *Mach effect*, and the combined front is known as the *Mach stem* (see Figure 4-4). The overpressure created by the Mach stem is typically twice that of the primary wave front alone. At first, the height of the Mach stem is short; but as the blast wave continues to move outward, the height increases steadily. However, as the height increases, the overpressure of the Mach stem decreases because of the continuous loss of energy and the increasing area of the advancing front. By 10 seconds after a detonation, the Mach stem has traveled about 5 km. Although the accompanying winds have deteriorated, they are still in excess of 150 mph. Thirty-seven seconds after the blast, the Mach stem has traveled just over 14 km (8.7 miles). Winds have diminished to below 40 mph, and the overpressure has decreased to about 1 psi (see Figure 4-5).

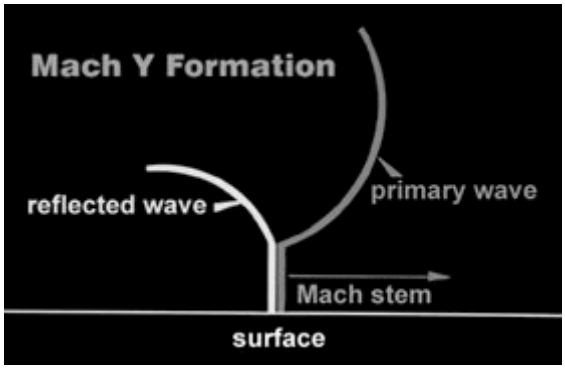

Figure 4-4. Mach wave formation.

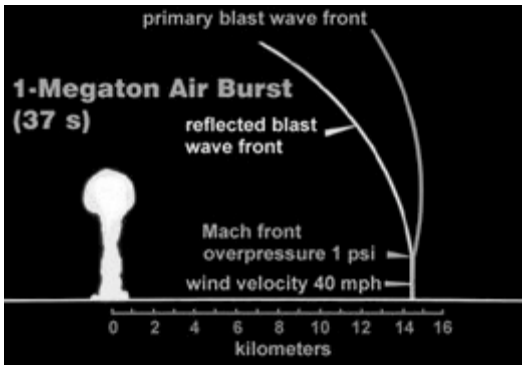

Figure 4-5. Blast wave 37 seconds after detonation.

4-6. Static and Dynamic Overpressures

Two distinct, though simultaneous phenomena are associated with the blast wave in air--static overpressure and dynamic pressures. Static overpressure (peak overpressure) is the sharp increase in pressure due to compression of the atmosphere. Dynamic pressures are the drag forces exerted by the strong transient blast winds associated with the movement of air required to form the blast wave.

a. Static overpressure, which is also called peak overpressure, is excess pressure, above atmospheric pressure, attributed to the explosion. If the peak overpressure is at least 0.5 psi, there will be some degree of material damage in most structures. Also, note in Table 4-2 that pressure of only 5 psi over atmospheric pressure is sufficient to destroy brick structures, flip automobiles, and knock down anchored poles.

Table, 4-2. Typical Overpressure Damage

PSI	DAMAGE
1	Windows shattered
2	Aluminum panels ripped off
3	Wall of 12-inch concrete shattered; parked aircraft destroyed
5	Brick houses destroyed; trucks overturned; telephone poles collapsed

b. Dynamic pressure, or drag force, consists of strong winds produced by differential pressure areas created by the blast wave. These forces are termed *dynamic* because they tend to push, tumble, and tear apart objects and cause their violent displacement. They occur in two phases, a compression phase and a negative phase (see Figure 4-6).

(1) In the first phase, compression occurs as the tremendous pressure created by the explosion pushes the blast wave front outward, away from the explosion. As the blast wave front moves away from the explosion, overpressure damage and dynamic pressure damage occur.

(2) In the second phase, the fireball continues to expand and rises rapidly, creating a region of low atmospheric pressure. The surrounding, high-pressure atmosphere rushes in to fill the low-pressure area. During this *negative phase*, strong winds called afterwinds travel toward the point of the explosion.

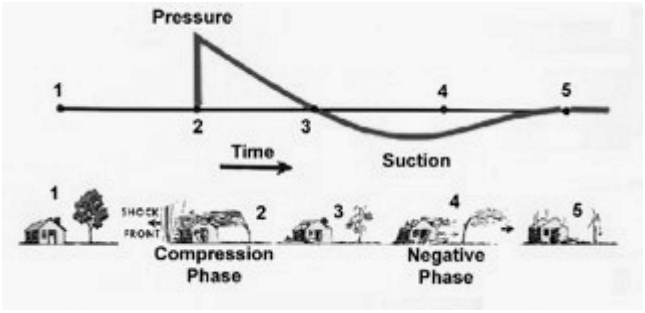

Figure 4-6. Phases of dynamic overpressure.

c. The destructive force associated with dynamic pressure is proportional to the square of their velocity. Equipment and personnel are relatively resistant to static overpressures but highly vulnerable to dynamic pressure. For example, military vehicles, from trucks to tanks, are most likely to suffer damage when pushed, overturned, and thrown about by blast winds. Likewise, blast winds are the cause of most blast injuries to personnel. Because of the violence of the winds associated with even low values of overpressure, mechanical injuries due to missiles sent into motion by the winds or to violent bodily translation will far outnumber direct blast injuries due to actual compression of the body.

d. The range for blast effects increases significantly with the explosive yield of the weapon. In a typical airburst, the values of overpressure and wind velocity noted above will prevail at a range of 0.7 km for 1 KT yield; 3.2 km for 100 KT; and 15.0 km for 10 MT. A surface burst results in the highest possible overpressures near ground zero. In such a burst, the shock front is hemispherical in form.

4-7. Blast Waves in Other Mediums

Underwater or water surface detonations will cause much greater subsurface shock waves. Because of water's density and relative incompressibility, the shock waves have very high peak overpressures and velocities of propagation. The peak overpressure at a distance of one km from

a 10 KT underwater burst is approximately 6,080 kilopascals (kPa) which equates to approximately 60 atmospheres (atm). The peak overpressure in air at the same distance from an airburst is only 111.4 kPa (1.1 atm). The resulting surface waves at this distance will be approximately 10 meters in height. The shock front will also travel at approximately five times the speed of the blast wave in air. Severe damage to naval vessels may result. Although the major portion of the shock energy is propagated in the water, a significant amount is also transferred through the surface as a typical air blast. This blast wave could probably be the principal source of damage to land targets if the explosion occurred in a coastal area.

Section III. THERMAL RADIATION

4-8. Formation of Thermal Radiation

a. Thermal radiation is emitted from the fireball within the first minute or less after detonation. It consists chiefly of radiation in the infrared, visible, and ultraviolet regions of the electromagnetic spectrum. Thermal exposure (measured in joules per unit area of exposed surface) will be less farther from the center of the explosion because the radiation is spread over a greater area and is attenuated in passing through the intervening air. Thermal radiation, infrared in particular, is primarily responsible for the majority of burns and fires associated with a nuclear detonation. The resultant burns can be classified as flash burns, those caused by direct exposure to the tremendous heat of the detonation, and flame burns, those that are a result of direct exposure to the fires created by the blast.

b. Immediately after an explosion, energy is emitted in the form of thermal radiation and as X-rays. These X-rays are absorbed within a few feet of air. The energy is then re-emitted and, due to certain phenomena occurring within the fireball, two pulses of secondary thermal radiation emission occurs (see Figure 4-7).

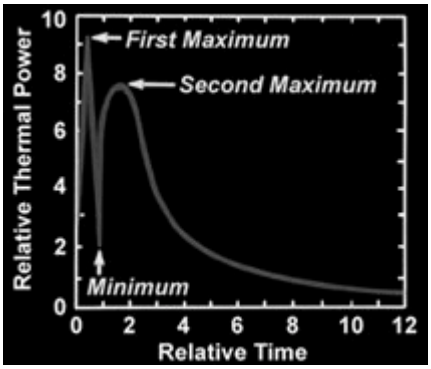

Figure 4-7. Thermal pulse production

(1) *First Pulse*. The first pulse lasts only about one tenth of a second. It consists of approximately one percent of the total thermal radiation release, and much of the thermal release is in the ultraviolet region of the spectrum. Although ultraviolet radiation can cause skin burns, it appears that this occurs only at ranges at which other thermal radiation effects are more serious. However, although this first pulse may be disregarded as a source of skin burns, it is capable of producing permanent or temporary effects on the eyes, especially if one is looking in the direction of the detonation at the time of the explosion.

(2) *Second Pulse*. The second pulse lasts for several seconds and carries about 99 percent of the total thermal radiation energy. Since the temperature is lower than that in the first pulse, most of the rays reaching the earth consist of visible and infrared light. This radiation is the main cause of various degrees of skin burns suffered by exposed individuals up to at least 12 miles.

4-9. Range and Intensity of Thermal Radiation

a. The fireball can be considered a point source of heat. The quantity of thermal radiation at any given point will vary approximately with the square of the distance from the explosion. Since thermal radiation travels in straight lines from the fireball, any opaque object interposed between the fireball and the target will act as a shield and provide significant protection from thermal radiation. If a significant amount of scattering is present, as is the case when dust and debris are in the air, thermal radiation will be received from all directions and shielding will be less effective.

b. The range of thermal effects increases markedly with weapon yield. The heat intensity at a given point will depend on the altitude and type of burst. In general, the thermal hazard is greatest in the case of a low altitude airburst. General thermal effects will be less for surface bursts and frequently nonexistent for subsurface bursts. In surface bursts, the ground or water absorbs a large part of the thermal energy. Shielding due to terrain and obscuration due to dust, moisture, and various gases in the air near the surface of the earth will tend to reduce the amount of thermal energy reaching a target.

4-10. Thermal Effects

a. When thermal radiation strikes an object, part will be reflected, part will be transmitted, and the rest will be absorbed. The fraction of the incident radiation that is absorbed depends on the nature and color of the material. A thin material may transmit a large part of the radiant energy striking it. A light colored object may reflect much of the incident radiation and thus escape damage. Thermal damage and injury is due to the absorption of large amounts of thermal energy within relatively short periods of time. The absorbed thermal radiation raises the temperature of the absorbing surface and results in scorching, charring, and possible ignition of combustible organic materials, such as wood, paper, fabrics, and so forth. If the target material is a poor thermal conductor, the absorbed energy is largely confined to a superficial layer of the material.

b. Actual ignition of materials exposed to thermal radiation is highly dependent on the duration of the thermal pulse (which is dependent on weapon yield) and the nature of the material, particularly its thickness and moisture content. The probability of significant fires, particularly firestorms, depends on the density of ignition points, the availability and condition of combustible material, wind, humidity, and the character of the surrounding area. Secondary fires started by blast wave effects, such as from upset stoves and furnaces, broken gas lines, and so forth, compound incendiary effects.

c. A firestorm is not a phenomenon peculiar to nuclear explosions. A firestorm is a massive conflagration that burns in upon itself with great ferocity because gale force winds blow in towards the center of the fire from all points of the compass. A firestorm due to conventional munitions started shortly after the incendiary bombing raids of Dresden and Tokyo during World War II. At Hiroshima, a tremendous firestorm developed within 20 minutes after the nuclear detonation. These intense conflagrations are also observed in large forest fires.

Section IV. NUCLEAR RADIATION AND FALLOUT

4-11. Initial Radiation

About five percent of the energy released in a nuclear airburst is transmitted in the form of initial neutron and gamma radiation. The neutrons result almost exclusively from the energy produced by fission and fusion reactions. The initial gamma radiation includes that arising from these reactions, as well as that from the decay of short-lived fission products. The intensity of the initial nuclear radiation decreases rapidly with distance from the point of burst. This is due to the spread of radiation over a larger area as it travels away from the explosion, and to absorption, scattering, and capture by the atmosphere. The character of the radiation received at a given location also varies with distance from the explosion. Near the point of the explosion, the neutron intensity is greater than the gamma intensity. With increasing distance the neutron-gamma ratio decreases. Ultimately, the neutron component of the initial radiation becomes negligible in comparison with the gamma component. The range for significant levels of initial radiation does not increase markedly with weapon yield. Therefore, the initial radiation becomes less of a hazard with increasing yield, as individuals close enough to be significantly irradiated, are killed by the blast and thermal effects. With larger weapons, above 50 KT, blast and thermal effects are so much greater in importance that prompt radiation effects can be ignored. The typical neutron dose-to-total dose ratios from a nuclear detonation based upon yield and range is shown in Table 4-3.

Table 4-3. Typical Neutron Dose-to-Total Dose Ratios

Yield (KT)	Neutron/Gamma	Neutron/Total Dose	Range (meters)
0.1	4.6	0.82	360
1.0	3.0	0.75	650
10.0	1.6	0.62	1040
100.0	0.47	0.32	1500
1000.0	0.042	0.04	2280
Assumptions: The height of the burst is 60 x $W^{1/3}$ meters, where W = yield in kilotons; air density is equal to 0.9, relative to sea level; fission only device.			

4-12. Residual Radiation

a. The residual radiation hazard from a nuclear explosion is in the form of neutron-induced activity and radioactive fallout. Residual ionizing radiation arises from a variety of sources. The first of these are the over 300 different fission products produced during detonation. These are intermediate weight isotopes which are formed when a heavy uranium or plutonium nucleus is split in a fission reaction. Many of these are radioactive with widely differing half-lives. Some fission products have half-lives which are very short; that is, fractions of a second. Other materials can be a hazard for months or years. Their principal mode of decay is by the emission of beta and gamma radiation. Approximately 60 grams of fission products are formed per KT of yield. The estimated activity of this quantity of fission products one minute after detonation is equal to that of 1.1 $x10^{21}$ Bq (30 million kilograms of radium) in equilibrium with its decay products.

b. Nuclear weapons are relatively inefficient in their use of fissionable material, and much of the uranium and plutonium is dispersed by the explosion without undergoing fission. Such unfissioned nuclear material decays primarily by the emission of alpha particles and is of relatively minor importance as long as it remains outside of the body. Also, the neutrons that are emitted as part of the initial nuclear radiation will cause activation of the weapon residues.

c. If atomic nuclei in soil, air, and water are exposed to neutron radiation and capture neutrons, they will, as a rule, become radioactive (neutron-induced activity) depending on their composition and distance from the burst. They then decay by emission of beta and gamma radiation over an extended period of time. For example, a small area around ground zero may become hazardous as a result of exposure of the minerals in the soil to initial neutron radiation. This is due principally to neutron capture by sodium, manganese, aluminum, and silicon in the soil. This is normally a negligible hazard because of the limited area involved. Tables 4-4 and 4-5 show the principle radionuclides induced in soil and the approximate yields of the principal nuclides from such weapons.

Table 4-4. Principal Radionuclides Induced in Soil

Isotope	Half-Life	Ci per Megaton
Sodium-24	15 hours	2.8×10^{11}
Phosphorus-32	14 days	1.92×10^{8}
Potassium-42	12 hours	3×10^{10}
Calcium-45	152 days	4.7×10^{7}
Moybdenum-56	2.6 hours	3.4×10^{11}
Iron-55	2.9 years	1.7×10^{7}
Iron-59	46 days	2.2×10^{6}

Table 4-5. Approximate Yields of the Principal Nuclides per Megaton of Fission

Nuclide	Half-Life	Mci
Strontium-89	53 days	20.0
Strontium-90	28 years	0.1
Zirconium-95	65 days	25.0
Ruthenium-103	40 days	18.5
Ruthenium-106	1 year	0.29
Iodine-131	8 days	125.0
Cesium-137	30 years	0.16
Cesium-131	1 year	39.0
Cesium-144	33 days	3.7

4-13. Fallout

After an airburst, the fission products, unfissioned nuclear material, and weapon residues that have been vaporized by the heat of the fireball will condense into a fine suspension of very small particles 0.01 to 20 micrometers in diameter. In a land or water surface burst, large amounts of earth or water will be vaporized by the heat of the fireball and drawn up into the radioactive cloud. This material will become radioactive when it condenses with fission products and other radioactive contaminants or if it has become neutron-activated. There will be large amounts of particles of less than 0.01 micrometer to several mm in diameter generated in a surface burst, in addition to very fine particles. These particles may be quickly drawn up into the stratosphere, especially if the explosive yield exceeds 10 KT. These materials will then be dispersed by atmospheric winds and will gradually settle to the earth's surface after weeks, months, and even years as fallout. The larger particles will not rise into the stratosphere and consequently will settle back to earth within about 24 hours as local fallout. Severe local fallout contamination can extend far beyond the blast and thermal effects, particularly in the case of high yield surface detonations. For subsurface bursts, there is an additional phenomenon present called *base surge*. The *base surge* is a cloud that rolls outward from the bottom of the column produced by a subsurface explosion.

a. The amount of activity associated with fallout depends on total energy yield, fusion fraction, weapon design, burst altitude, nature of the surface at ground zero,

meteorological conditions, and time after the explosion. Due to the variability of several of these factors, particularly meteorological conditions, the portion of activity due to fallout is not figured into the weapon's total energy yield. Only the blast, thermal, and initial radiation are considered.

(1) A fission reaction results in two fission fragments of varying elemental form. For each KT of fission energy yield, roughly 2 ounces of fission product atoms are formed. During fission, the nucleus of the fuel atom can split in about 40 different ways, thereby producing more than 80 fission fragments consisting of more than 300 isotopes of 36 elements, all of them radioactive. Their atomic numbers range from 30 (zinc-72) to 64 (gadolinium-158).

(2) The vast majority of the fission products fall into two groups: The lighter group, peaking near mass number 90, and the heavier group, near 137. These two groups represent the two most significant long-term fallout hazards: Strontium-90 and cesium-137. Similar groups would result from fissioning plutonium. Also, iodine-131 presents a hazard, but since it has a relatively short half-life (~ 8 days), it is considered a short-term hazard.

b. In some scenarios, if certain atmospheric conditions are present, and if large yield weapons are used, then the fallout could be worldwide. The radiobiological hazard of worldwide fallout is essentially a long term one due to the potential accumulation of long-lived radioisotopes, such as strontium-90 and cesium-137 in the body as a result of ingestion of foods or water that have incorporated these radioactive materials. This hazard is much less serious than those that are associated with local fallout that is of much greater immediate operational concern.

c. Whenever individuals remain in a radiologically contaminated area, such contamination will lead to an immediate external radiation exposure as well as a possible later internal hazard due to inhalation and ingestion of radio contaminates. In severe cases of fallout contamination, lethal doses (LDs) of external radiation may be incurred if protective or evasive measures are not undertaken. In cases of water surface (and shallow underwater) bursts, the particles tend to be lighter and smaller. This produces less local fallout by extending the spread of contamination over a greater area. The particles contain mostly sea salts with some water; these can have a cloud seeding affect, causing local rain out and areas of high local fallout.

d. Meteorological conditions will greatly influence fallout, particularly local fallout. Atmospheric winds are able to distribute fallout over large areas. For example, as a result of a surface burst of a 15 MT thermonuclear device at Bikini Atoll on March 1, 1954, a roughly cigar-shaped area of the Pacific extending over 500 km downwind and varying in width to a maximum of 100 km was severely contaminated. Snow and rain, especially if they come from considerable heights, will accelerate local fallout. Under special meteorological conditions, such as a local rain shower originating above the radioactive cloud, limited areas of heavy contamination may be formed.

e. *Scavenging* refers to processes that increase the rate at which radioactivity is removed from the fallout cloud and deposited on the earth's surface. Even in the case of an airburst, which does not usually produce early fallout, rainout or washout can cause significant contamination on the ground as a result of scavenging of radioactive debris. This contamination

is typically found in concentrated hotspots created between ridges in the earth's surface or wherever rainwater collects. While the process by which vaporized fission fragments condense on dirt and debris is a form of scavenging, precipitation scavenging is of primary interest. Precipitation scavenging is the process in which rain or snow falls through the fallout cloud and carries contaminated particles down with it. Precipitation scavenging occurs in two forms--rainout and washout.

(1) *Rainout*. Rainout occurs when a rain cloud forms within the fallout cloud. The rate of rainfall has little influence on the effectiveness of rainout because scavenging takes place as raindrops form in the rain cloud.

(2) *Washout*. Washout occurs when the rain cloud forms above the fallout cloud. The strength of the rain and the length of time the radioactive cloud is *washed* markedly affect the percentage of radioactivity scavenged. Washout is greatly affected by the speed and direction of the rain cloud relative to the fallout cloud as well as to when and if the rain cloud intercepts the fallout cloud. Evidence indicates that washout is far less effective than rainout.

CHAPTER 5

BLAST AND THERMAL MEDICAL EFFECTS OF A NUCLEAR EXPLOSION: DIAGNOSIS, TREATMENT, AND PROGNOSIS

Section I. BLAST INJURY

5-1. General

There are two basic types of blast forces which occur simultaneously in a nuclear detonation blast wave; these are direct blast wave overpressure forces, measured in terms of atmospheres of overpressure; and indirect blast wind drag forces, normally measured in the velocities of the wind which cause them. The most important blast effects, insofar as production of casualties requiring medical treatment is concerned, will be those due to the blast wind drag forces. Direct overpressure effects do not extend out as far from the point of detonation and are frequently masked by drag force effects as well as by thermal effects. However, direct blast effects can contribute significantly to the immediate deaths and injuries sustained close to the point of detonation and, therefore, do constitute an important total casualty producing effect. Personnel in fortifications or heavy armored vehicles, such as tanks, who are protected from radiation and thermal and blast wind effects, may be subjected to complex patterns of direct overpressures since blast waves can enter such structures and be reflected and reinforced within them.

5-2. Direct Blast Injury

a. When a blast wave hits a target, the nature and probability of damage will depend upon a number of variables in the characteristics of the blast wave and of the target. Important variables of the blast wave include--

- The rate of pressure rises at the blast wave front.
- The magnitude of the peak overpressure.
- The duration of the blast wave.

Important variables of the target include size, mass, density, resistance to deformity, and so forth. If the target is human, then additional factors, such as age, physical condition, and the presence of disease or other injury, become important.

b. When the blast wave acts directly upon a resilient target such as the human body, rapid compression and decompression result in transmission of pressure waves through the tissues. These waves can be quite severe and will result in damage primarily at junctions between tissues of different densities (bone and muscle) or at the interface between tissue and air spaces. Lung tissue and the GI system, both of which contain air, are particularly susceptible to injury. The resulting tissue disruptions can lead to severe hemorrhage or to air embolism, either

of which can be rapidly fatal. Perforation of the eardrums would be a common but a minor blast injury.

c. The range of overpressures associated with lethality can vary greatly. It has been estimated that overpressures as low as 193 kPa (1.9 atm) can be lethal, but that survival is possible with overpressures as high as 262 kPa (2.5 atm). A typical range of probability of lethality with variation in overpressure is summarized in Table 5-1. These are rough estimates based on selected experimental data, and there are some differences between these figures and data based upon other experimental work. In addition, these numbers apply only to unreinforced, unreflected blast waves. When blast waves are complicated by reinforcement and reflection, estimation or measurement of the overpressures associated with specific injuries becomes quite complex. The significant thing shown by the data in Table 5-1 is that the human body is remarkably resistant to static overpressure, particularly when compared with rigid structures such as buildings. Shattering of an unreinforced cinder block panel, for example, will occur at 10.1 to 20.2 kPa (0.1 to 0.2 atm).

Table 5-1. Range of Lethality at Peak Overpressure

Lethality (approximate %)	**Peak Overpressure (kPa)**
1	160 – 230
50	230 – 400
100	400+

d. Overpressures considerably lower than those listed in Table 5-1 will cause injuries which are not lethal. Lung damage and eardrum rupture are two useful biomedical parameters to use as examples, since one is a relatively serious injury, usually requiring hospitalization even if not lethal, while the other is a minor injury, often requiring no treatment at all. The threshold level of overpressure (which is estimated to cause lung damage) is about 68.9 kPa for a simple unreinforced, unreflected blast wave. There will be considerable variation in this value with differing conditions of exposure. The threshold value for eardrum rupture is probably around 22 kPa (0.2 atm) and that overpressure associated with a 50 percent probability of eardrum rupture ranges from 90 to 130 kPa (0.9 to 1.2 atm).

e. From the examples above, it can be seen that casualties requiring medical treatment from direct blast effects could, theoretically, be produced by overpressures greater than 70 kPa. However, direct blast injuries will not occur by themselves; in general, other effects, such as indirect blast injuries and thermal injuries are so severe at the ranges associated with these overpressures that patients with direct blast injuries will comprise a very small part of the patient load.

5-3. Indirect Blast Wind Drag Forces

a. Blast Winds. The drag forces of blast winds are proportional to the velocities and duration times of those winds, which in turn vary with distance from the point of detonation,

yield of the weapon, and altitude of the burst. These winds are relatively short in duration but are extremely severe. The winds can be much greater than the strongest hurricane winds and may reach several hundred km per hour. Indirect blast injuries will occur as translocation and deceleration injuries. Casualties will be thrown against immobile objects and impaled by flying debris; therefore, solid organ, extremity, and head injuries will be commonplace.

b. *Probability of Indirect Blast Injury.* The distance from the point of detonation at which severe indirect injury will occur is considerably greater than that for serious direct blast injuries. It is difficult to give precise ranges at which these indirect injuries are likely to occur because of the marked effect of variations in the environment. However, the peak overpressure range of approximately 20.3 kPa (0.2 atm) is a reasonable reference distance at which the probability of serious indirect injury is high. Injuries can occur at greater ranges and casualties will be generated at greater ranges, but not consistently.

5-4. Missile Injury

a. The probability of injury from a missile depends upon the number of missiles generated, their individual kinetic energies, their shape, and their composition. The number of missiles that can be generated by the blast winds depends to some extent upon the environment, that is, different terrain types will have different quantities of material available for missile production. However, the drag forces of the blast winds produced by nuclear detonations are so great that almost any form of vegetation or structure, if present, will be broken apart or fragmented into a variety of missiles. Single missile injuries will be rare and multiple, varied missile injuries will be common. Table 5-2 gives an indication of the ranges significant missile injuries (either potentially fatal or requiring surgical attention) would be expected.

Table 5- 2. Ranges for Different Probabilities of Injury from Small Missiles

Yield (kt)	Range (km) for Probability of Serious Injury		
	1% probability	50% probability	99% probability
1	0.28	0.22	0.17
10	0.73	0.57	0.44
20	0.98	0.76	0.58
50	1.4	1.1	0.84
100	1.9	1.5	1.1
200	2.5	1.9	1.5
500	3.6	2.7	2.1
1000	4.8	3.6	2.7

b. The kinetic energy, composition, and shape of missiles involve a detailed discussion of complex ballistics that is beyond the scope of this manual. Objects cannot be made to go faster than the winds themselves; therefore these missiles will be low velocity in nature. None will be high velocity, such as is produced with small arms fire. The weight or mass of an object and the duration times of the winds determine whether or not that object will be

accelerated maximally. Light objects will be accelerated rapidly up to the maximum possible velocity, whereas heavy objects may not be. The probability of a penetrating injury increases with increasing velocity, particularly for small, sharp missiles such as glass fragments. Heavier objects require higher kinetic energies to penetrate, therefore, heavy blunt missiles will not ordinarily penetrate the body but can result in significant injury, particularly fractures.

5-5. Crush and Translational Injuries

a. The drag forces of the blast winds are strong enough to displace even large objects, such as vehicles, or to cause the collapse of large structures, such as buildings. These events can result in very serious crush injuries. These injuries would be comparable to those seen in earthquakes and bombings. A human body can itself become a missile and be displaced a variable distance and at variable velocities depending upon the intensity of the drag forces and the nature of the environment. The resulting injuries sustained are termed translational injuries. The probability and the severity of injury are functions of the velocity of the human body at the time of impact. If a representative displacement distance of 3.0 meters is assumed, the impact velocities that would be associated with various degrees of injury can be calculated. These are shown in Table 5-3. The table shows terminal or impact velocities associated with significant but nonlethal blunt injury. It also shows those velocities that are associated with a probability of lethality. The velocities in Table 5-3 can be equated against yield, and the ranges at which such velocities would be found can be calculated. These are shown in Table 5-4.

Table 5-3. Translational Injuries

Velocity* ($m\ sec^{-1}$)	Probability of Blunt Injuries & Fractures	Probability of Fatal Injuries
2.6	>1%	-
6.6	~50%	>1%
17.0	99%	~50%
44.5	-	99%

*Velocities are based on solid impact with a non-yielding surface.

Table 5-4. Ranges for Selected Impact Velocities of a 70-kg Human Body Displaced by Blast Wind Drag Forces for Different Yield Weapons

Weapon Yield (kt)	Velocities* ($m\ sec^{-1}$) 2.6	6.6	17.0
	Range (km)		
1	0.38	0.27	0.19
10	1.0	0.75	0.53
20	1.3	0.99	0.71
50	1.9	1.4	1.0
100	2.5	1.9	1.4
200	3.2	2.5	1.9
500	4.6	3.6	2.7
1000	5.9	4.8	3.6

5-6. Diagnosis

The blast injuries caused by nuclear weapons will frequently be complicated by associated thermal and/or radiation injuries. Also, the number of casualties produced at any one time in a given area will be much greater for nuclear weapons than for conventional weapons. The diagnosis of blast injuries is generally not difficult unless there is unrecognized internal injury with slow hemorrhage since missile injuries will predominate. About half of the patients seen will have wounds of their extremities. The thorax, abdomen, and head will be involved about equally. Missile injuries of the thorax, neck, and the head will be responsible for a large percentage of deaths because these types of injuries have a high probability of immediate fatality. The missile injuries caused by nuclear weapons will, in general, be of the low velocity type, and surprisingly, severe injuries may even be survivable since extensive soft tissue cavitation would not be a factor. These injuries can occur with or without perforating wounds of the abdomen or the chest.

5-7. First Aid

a. Blast injuries require immediate attention at the individual level. The basic lifesaving steps of clear the airway/restore breathing, stop the bleeding, protect the wound, and treat/prevent shock apply to first-aid measures for all injuries. Therefore, the immediate concern is checking casualties for an inadequate airway, lack of breathing or lack of heartbeat, and excessive loss of blood because these problems may become life threatening. A casualty without a clear airway or who is not breathing may die from lack of oxygen. Excessive loss of blood may lead to shock, and shock can lead to death; therefore, US personnel must act immediately to control the loss of blood. All wounds are considered to be contaminated because of infection-producing organisms (germs) and radiological material due to fallout. That a wound is contaminated does not lessen the importance of protecting it from further contamination. Personnel must dress and bandage a wound as soon as possible to prevent further contamination.

b. Missile, crush, and translational injuries are generally manifested as wounds of the head, neck, face, chest stomach, and fractures. A brief discussion of the first aid for each of these wounds is discussed below. For a detailed discussion on first aid for these wounds, see FM 21-11.

(1) *Head injuries*. A head injury may consist of one or a combination of the following conditions: a concussion, a cut or bruise of the scalp, or a fracture of the skull with injury to the brain and the blood vessels of the scalp. The damage can range from a minor cut on the scalp to a severe brain injury that rapidly causes death. Most head injuries lie somewhere between the two extremes. Usually, serious skull fractures and brain injuries occur together; however, it is possible to receive a serious brain injury without a skull fracture. The brain is a very delicate organ; when it is injured, the casualty may vomit, become sleepy, suffer paralysis, or lose consciousness and slip into a coma. All severe head injuries are potentially life threatening. For recovery and return to normal function, casualties require proper first aid as a vital first step. Also, a person that has an injury above the collarbone or a head injury resulting in an unconscious state should be suspected of having a neck or head injury with spinal cord

damage. A lack of responses to stimuli, stomach distention (enlargement), or penile erection may indicate spinal cord injury. General first-aid procedures include the following.

- Perform basic lifesaving measures.

- Place a dressing over the wound; for a head injury, **do not** attempt to clean the wound.

- **Do not** attempt to put unnecessary pressure on the wound; **do not** apply a pressure dressing or attempt to push any brain matter back into the head (skull).

- Keep the casualty warm.

- **Do not** attempt to remove a protruding object from the head.

- **Do not** give the casualty anything to eat or drink.

- **Do not** move the casualty if you suspect he has sustained a neck, spine, or severe, head injury.

(2) *Face and neck injuries*. Soft tissue injuries of the face and scalp are common. Abrasions (scrapes) of the skin cause no serious problems. Contusions (injury without a break in the skin) usually cause swelling. A contusion of the scalp looks and feels like a lump. Laceration (cut) and avulsion (torn away tissue) injuries are also common. Avulsions are frequently caused when a sharp blow separates the scalp from the skull beneath it. Because the face and scalp are richly supplied with blood vessels (arteries and veins), wounds of these areas usually bleed heavily. Neck injuries may result in heavy bleeding. Apply manual pressure above and below the injury and attempt to control the bleeding and apply a dressing to the wound. Always evaluate the casualty for a possible neck fracture/spinal cord injury; if suspected, seek medical treatment immediately. General first-aid procedures include--

- Performing basic lifesaving measures.

- Clearing the casualty's airway (mouth) with your fingers, remove any blood, mucus, pieces of broken teeth or bone, or bits of flesh, as well as any dentures.

- Controlling any bleeding, especially bleeding that obstructs the airway. Do this by applying direct pressure over a first-aid dressing or by applying pressure at specific pressure points on the face, scalp, or temple. **Do not** apply too much pressure to the scalp if a skull fracture is suspected.

- Placing the casualty in a comfortable sitting position and having him lean forward with his head tilted slightly down to permit free drainage if the casualty is bleeding from the mouth (or has other drainage, such as mucus, vomitus, or so forth) and is conscious.

- Not using the sitting position if it would be harmful to the casualty because of other injuries; if the casualty is unconscious, placing him on his side.

- If there is a suspected injury to the neck or spine, immobilizing the head before turning the casualty on his side.

(3) *Eye injuries*. Injuries of the eye may be quite severe. Cuts of the eyelids can appear to be very serious, but if the eyeball is not involved, a person's vision usually will not be damaged. However, lacerations (cuts) of the eyeball can cause permanent damage or loss of sight. Lacerated eyelids may bleed heavily, but bleeding usually stops quickly. Cover the injured eye with a sterile dressing, but do not put pressure on the wound because of possible injury to the eyeball. Handle torn eyelids very carefully to prevent further injury; place any detached pieces of the eyelid on a clean bandage or dressing and immediately send them with the casualty to the MTF. Lacerations or cuts to the eyeball may cause serious and permanent eye damage. Cover the injury with a loose sterile dressing, and do not put pressure on the eyeball because additional damage may occur. An important point to remember is that when one eyeball is injured, both eyes should be immobilized by applying a bandage to both eyes. Because the eyes move together, covering both will lessen the chances of further damage to the injured eye.

(4) *Chest wounds*. A casualty with a chest injury normally has pain in the chest or shoulder area and he may have difficulty with his breathing. His chest may not rise normally when he breathes, and the injury may cause the casualty to cough up blood and to have a rapid or a weak heartbeat. A casualty with an open chest wound has a punctured chest wall. The sucking sound heard when he breathes is caused by air leaking into his chest cavity. This particular type of wound is dangerous and will collapse the injured lung. Breathing becomes difficult for the casualty because the wound is open, and the person's life depends upon how quickly the wound can be made airtight. General first-aid procedures include--

- Perform basic lifesaving measures.

- Cutting or removing the casualty's clothing to expose the entire area of the wound. **Do not** remove clothing that is stuck to the wound because additional injury may result; **do not** attempt to clean the wound.

- Using the field dressing plastic wrapper to create an airtight seal by placing the inside surface of the plastic wrapper directly over the wound *when the casualty exhales* and holding it in place. If the plastic wrapper is not available, or if an additional wound needs to be treated, cellophane, foil, the casualty's poncho, or similar material may be used. The covering should be wide enough to extend 2 inches or more beyond the edges of the wound in all directions.

- Applying the field dressing by placing the white side of the dressing on the plastic wrapper covering the wound and securing it to the casualty.

(5) *Abdominal wounds.* The most serious abdominal wound is one in which an object penetrates the abdominal wall and pierces internal organs or large blood vessels. In

these instances, bleeding may be severe and death can occur rapidly. General first-aid procedures include--

- Performing basic lifesaving measures.

- Placing and maintaining the casualty on his back with his knees in an upright (flexed) position. The knees-up position helps relieve pain, assists in the treatment of shock, prevents further exposure of the bowel (intestines) or abdominal organs, and helps relieve abdominal pressure by allowing the abdominal muscles to relax.

- Removing the casualty's loose clothing to expose the wound. **Do not** attempt to remove clothing that is stuck to the wound as it may cause further injury.

- Gently picking up any organs which may be on the ground with a clean, dry dressing or with the cleanest available material and placing the organs on top of the casualty's abdomen. **Do not--**

  - Probe, clean, or try to remove any foreign object from the abdomen.

  - Touch with bare hands any exposed organs.

  - Push organs back inside the body.

- Applying the field dressing.

(6) *Fractures.* A fracture is any break in the continuity of a bone. Fractures can cause total disability, or in some cases, death. On the other hand, they can most often be treated so there is complete recovery. A great deal depends upon the first aid the individual receives before he is moved, which includes immobilizing the fractured part in addition to applying lifesaving measures. The basic splinting principle is to immobilize the joints above and below any fracture. Indications of a fracture are deformity, tenderness, swelling, pain, inability to move the injured part, protruding bone, bleeding, or discolored skin at the injury site. A sharp pain when the individual attempts to move the part is also a sign of a fracture. DO NOT encourage the casualty to move the injured part in order to identify a fracture since such movement could cause further damage to surrounding tissues and promote shock. If unsure whether a bone is fractured, treat the injury as a fracture. First-aid procedures include the following:

- Monitor the casualty for development of conditions which may require you to perform necessary basic lifesaving measures. These measures include clearing the airway, performing rescue breathing, preventing shock, and/or controlling bleeding.

- Gather whatever splinting materials are available; such as splints, boards, branches, or poles, padding, improvised cravats, and/or bandages. Ensure that splints are long enough to immobilize the joint above and below the suspected fracture.

- Unless there is immediate life-threatening danger, such as a fire or an explosion, do not move the casualty with a suspected back or neck injury. Improper movement may cause permanent paralysis or death. In a chemical environment, do not remove any protective clothing; apply the dressing/splint over the clothing.

- Locate the site of the suspected fracture; prepare the casualty for splinting by reassuring the him, by loosening any tight or binding clothing, and by removing all the jewelry from the casualty. Boots should not be removed from the casualty unless they are needed to stabilize a neck injury, or if there is actual bleeding from the foot.

- Pad the splints where they touch any bony part of the body, such as the elbow, wrist, knee, ankle, crotch, or armpit; this prevents excessive pressure to the injured area.

- Check the circulation below the site of the injury; also, check the temperature of the injured extremity and question the casualty about the presence of numbness, tightness, cold, or tingling sensations. Casualties with impaired circulation should be evacuated by medical personnel and treated as soon as possible since this may prevent possible loss of the limb.

- Splint the fracture(s) in the position found; do not attempt to reposition or straighten the injury. If bones are protruding (sticking out), do not attempt to push them back under the skin. Apply dressings to protect the area.

- Place one splint on each side of the arm or leg and ensure that the splints reach beyond the joints above and below the fracture.

- Secure each splint in place above and below the fracture site with improvised (or actual) cravats. Improvised cravats, such as strips of cloth, belts, or whatever else you have, may be used. With minimal motion to the injured areas, place and tie the splints with the bandages. Tie all knots on the splint away from the casualty and do not tie cravats directly over suspected fracture/dislocation site.

- Check to ensure that bandages are tight enough to securely hold splinting materials in place, but not so tight that circulation is impaired. Recheck the circulation after application of the splint. This is to ensure that the bandages holding the splint in place have not been tied too tightly.

5-8. Treatment

The treatment of blast injuries, whether combined with other injuries or not, is best managed by applying accepted principles of combat surgery. Treatment is divided into following four basic phases:

a. Resuscitative Phase. Lifesaving resuscitative measures designed to prepare the patient for definitive surgical treatment come first. These include the establishment of the airway assuring the adequacy of respiration, replacement of lost blood and fluids, and splinting of possible fractures, particularly those involving the cervical vertebrae. Some resuscitative measures must be started prior to evacuation from the battlefield, particularly if ground transportation is used rather than helicopter evacuation.

b. Surgical Phase.

(1) Definitive surgery should be done after resuscitative measures have been used to improve the patient's condition in order to minimize the risk of surgery and anesthesia. Occasionally, lifesaving surgery must be done without delay, but normally there is time to prepare patients for surgery if they have survived long enough to reach a treatment facility.

(2) Blast casualties will require evaluation for acute trauma in accordance with advanced trauma management standard therapies. Pneumothorax secondary to both direct and indirect trauma and pneumoperitoneum can be treated by appropriate surgical intervention. Subsequent delayed sequaelae such as pulmonary failure may result from severe barotrauma. Response to therapy will be complicated by any radiation exposure due to immune system compromise and delayed wound healing. Open wounds will require thorough debridement and removal of all contaminants including radioactive debris. Suspected fractures should be splinted. Spinal injuries resulting from either translational injury or blunt force trauma should be treated with immobilization. In the presence of traumatic injury, hypotension must be considered to be due to hypovolemia and not to concomitant head or radiological injury. Surgical priorities for acute or life-threatening injury must precede any treatment priority for associated radiation injury. Treatment of tympanic membrane rupture can be delayed.

c. Recovery Phase. In the immediate postoperative period, patients require minimal movement. Transportation to other facilities should be delayed until the patient's condition has stabilized.

d. Convalescent Phase. Patients in this phase of treatment should be evacuated back to specialized convalescent facilities in order to keep the patient load of supporting hospitals as low as possible. Many injuries may require a prolonged recovery period before the individual has recovered to the point where he/she can resume their duties. Both the convalescent and recovery phases will be more protracted with the addition of a radiation injury.

Section II. THERMAL INJURY

5-9. Thermal Effects

Many burn casualties may occur as a result of incendiary attacks on cities and military personnel in the field during conventional warfare. However, in a nuclear environment, thermal burns will be the most common injuries, subsequent to both the thermal pulse, and the fires it ignites. The

thermal radiation emitted by a nuclear detonation causes burns in two ways, by direct absorption of the thermal energy through exposed surfaces (flash burns) or by the indirect action of fires caused in the environment (flame burns). The relative importance of these two processes will depend upon the nature of the environment. If a nuclear weapon detonation occurs in easily flammable surroundings, indirect flame burns could possibly outnumber all other types of injury. Because of the complexity of burn treatment and the increased logistical requirements associated with the management of burns, they will constitute the most difficult problem faced by the medical service.

a. Flash (Thermal Pulse) Burns.

(1) Since the thermal pulse is direct infrared, burn patterns will be dictated by spatial relationships and clothing pattern absorption. Exposed skin will absorb the infrared in a variable pattern and the victim will be burned on the side facing the explosion. Light colors will reflect the infrared while dark portions of clothing will absorb it and cause pattern burns. Figure 5-1 shows a Nagasaki woman with flash burns in the pattern of the kimono she was wearing at the time of the blast. The light-colored fabric reflected the thermal radiation whereas the dark-colored fabric absorbed it and became hot, causing contact burns on the skin immediately beneath the fabric. Medical records indicate that, in some cases, dark-colored clothing actually burst into flames and ignited the undergarments, causing flame burns. Persons shaded from the direct light of the blast were protected. However, the attenuating effect of even a heavy cloud cover is surprisingly small. Close to the fireball, the thermal output is so great that everything is incinerated. Obviously, immediate lethality would be 100 percent within this range. The actual range out to which overall lethality would be 100 percent will vary with yield, position of burst, weather, the environment and how soon those burned can receive medical care. The mortality rate among the severely burned is much greater without early resuscitative treatment. Table 5-5 shows the probabilities of radiation or thermal burns based upon the yield of the weapon.

Figure 5-1. Thermal flash burns in the pattern of dark clothing.

Table 5-5. Probability of Radiation or Thermal Burns.

Yield of Weapon	**1 KT**	**10 KT**	**100 KT**	**1 MT**	**10 MT**
Range (km)	0.78	2.1	4.8	9.1	14.5

for production of second-degree burns on exposed skin.					
Duration of thermal pulse in seconds.	0.12	0.32	0.9	2.4	6.4

(2) While most thermal injury predictions refer to exposed skin, it is important to remember the protection from burns that can be achieved with clothing. That protection, however, is not absolute. At temperatures below those required to ignite clothing, it is possible to transfer sufficient thermal energy across clothing to the skin to produce flash burns. The amount of heat energy conducted across clothing is a function of the energy absorbed by, and the thermal conducting properties of the clothing. It will also be a function of whether the clothing is tight fitting or loose. Clothing significantly reduces the effective range producing partial thickness burns, thus affording significant protection against thermal flash burns.

b. Flame Burns. Firestorm and secondary fires will cause typical flame burns, but they will be compounded by closed space fire injuries. Patients with toxic gas injury from burning plastics and other material, superheated air inhalation burns, steam burns from ruptured pipes and all other large conflagration-type injuries will require treatment. The injury distribution will be determined by the nature of the target. Indirect or flame burns result from exposure to fires caused by the thermal effects in the environment, particularly from ignition of clothing. This could be the predominant cause of burns depending on the number of and characteristics of flammable objects in an environment. This is particularly true for the large yield weapons, which can cause conflagrations and fire storms over extensive areas. Complications arise in the treatment of skin burns created, in part, from melting of man-made fibers. Clothing made of natural fibers, or flame resistant clothing (for example, Nomex) should be worn next to the skin. The probability of flame burns cannot be quantified with range as well as can that of flash burns. The variables of environmental flammability are too great to allow prediction of either incidence or severity. Depending on the flammability of the material, blast winds can either extinguish or fan the burning material. The burns themselves will be far less uniform in degree and will not be limited to exposed surfaces. For example, the respiratory system may be exposed to the effects of hot gases, and respiratory system burns are associated with severe morbidity and high mortality rates. Early endotracheal intubation is advisable whenever airway burns are suspected.

c. Eye Injuries. Sudden exposures to high-intensity sources of visible light and infrared radiation can cause eye injury, specifically to the chorioretinal areas. Although the most common source of thermal energy injury to the eye is from directly viewing the sun, other sources of luminance including nuclear explosions can cause eye injuries. Factors that determine the extent of eye injury include pupil dilation, spectral transmission through the ocular media, spectral absorption by the retina and choroid, length of time of exposure, and the size and quality of the image. Direct vision optical equipment such as binoculars will increase the likelihood of

damage. Night vision devices (NVDs) electronically amplify the ambient light, and they do detect infrared energy, the major component of the thermal pulse. However, most NVDs automatically shutdown when an intense burst of energy hits the device. Eye injury is due not only to thermal energy but also to photochemical reactions that occur within the retina with light wavelengths in the range of 400 to 500 ηm. Only the use of lead-lanthanum-zirconium-titanium goggles will fully protect the eyes from this type of injury. Since most personnel will not have access to these goggles, there will be numerous eye injuries which will require treatment.

(1) *Flash blindness*. Flash blindness occurs with sudden peripheral visual observation of a brilliant flash of intense light energy; for example, a fireball. This is a temporary condition that results from a depletion of photopigment from the retinal receptors. The duration of flash blindness can last several seconds when the exposure occurs during daylight. The blindness will then be followed by a darkened after-image that lasts for several minutes. At night, flash blindness can last for up to 30 minutes and may occur km from the blast (see Figure 5-2).

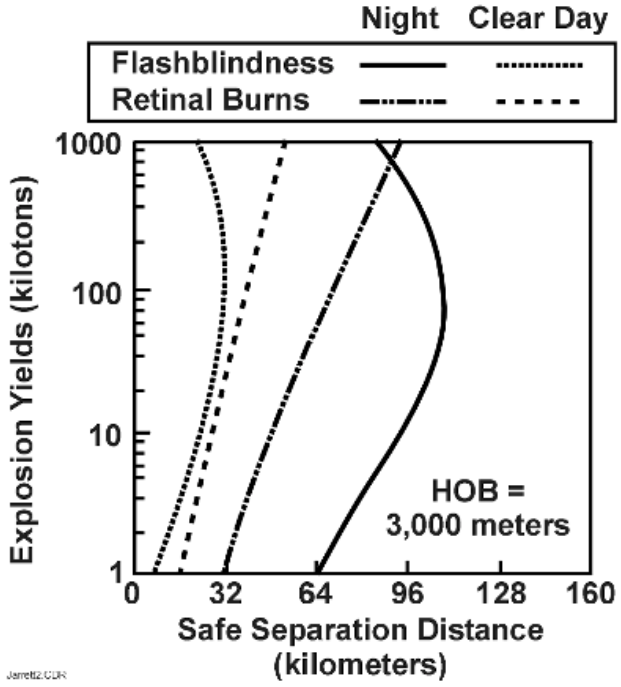

Figure 5-2, Flash blindness and retinal burn safe separation.

(2) *Retinal burns.* Direct observation of a brilliant flash of light in the wavelengths of 400 to 1,400 ηm can cause macular-retinal burns. Burns of the macula will result in permanent scarring with resultant loss in visual acuity. Burns of the peripheral regions of the retina will produce scotomas, blind spots, but overall visual acuity will be less impaired. These burns can occur at distances of several miles under optimal conditions.

5-10. Examples of Blast and Thermal Scenarios

a. Injury Risk. As shown in Figure 5-3, the blast and thermal effects in a nuclear detonation are greater than the nuclear radiation effects except at extremely low yields including small weapons that produce only localized effects. For weapons of at least 1 KT, thermal effects and blast wave effects dominate.

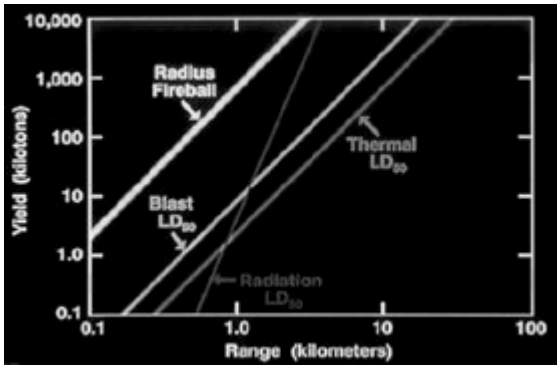

Figure 5-3. Blast and thermal effects versus yield.

b. Detonation Scenario: One Kiloton Device. Assume that a 1 KT nuclear device is detonated atop the Washington Monument in downtown Washington, DC. As shown in Figure 5-4, the monument is south of the White House and just west of the Lincoln Memorial. To the west of the monument is The Mall, comprised of the Smithsonian Institute buildings and the Capitol Building. The monument is slightly taller than 555 feet.

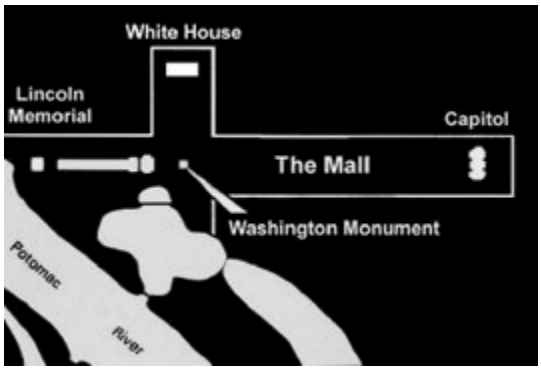

Figure 5-4, Location of a One Kiloton Device.

(1) *Static overpressure damage*. The static overpressure from the detonation of a 1 KT device atop the Washington Monument would kill at least 50 percent of the population within 150 meters of the monument. Lung damage would occur in 50 percent of the population within 300 meters. The eardrums would rupture in 50 percent of the population up to 700 meters away. Within the 700 meters, brick structures would likely be destroyed by the peak

overpressure of 5 PSI. The White House would probably remain standing, but windows would be shattered (see figure 5-5).

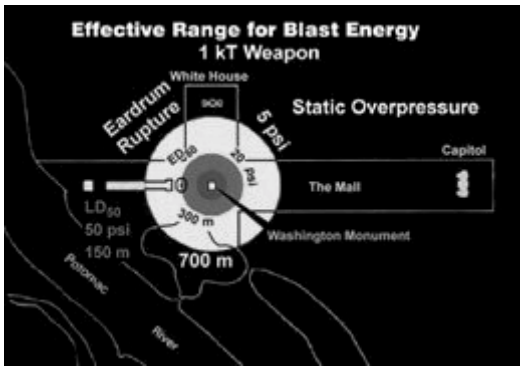

Figure 5-5. One kiloton weapon static overpressure ranges.

(2) *Dynamic overpressure damage*. Fifty percent of the population within 350 meters of the detonation would be killed by the force of the dynamic overpressure of the blast wave, or wind. LD_{50} for impact is 11 meters per second. Within 550 meters of the monument, people would suffer some injury, perhaps broken bones. At 750 meters, fragments of shattered glass would fly through the air at 55 meters per second, sufficient to cause severe lacerations and life-threatening penetrating wounds (see Figure 5-6).

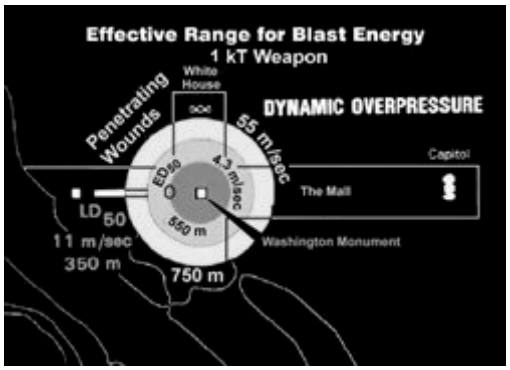

Figure 5-6, One kiloton weapon dynamic overpressure ranges.

(3) *Thermal radiation damage*. Out to 700 meters, exposure to thermal radiation would be around seven calories per square centimeter, enough to produce third-degree burns. Out to 800 meters, exposed persons would receive second-degree burns. As far out as 1,200 meters, one could expect first-degree burns. These injuries are in addition to any flame burns from ignited combustibles (see Figure 5-7).

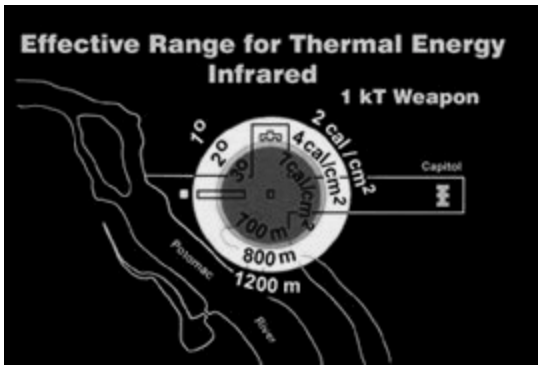

Figure 5-7. One kiloton weapon thermal energy ranges.

(4) *Thermal radiation: one-megaton device.* In contrast, Figure 5-8 illustrates the burn distribution profile expected with a 1 MT weapon.

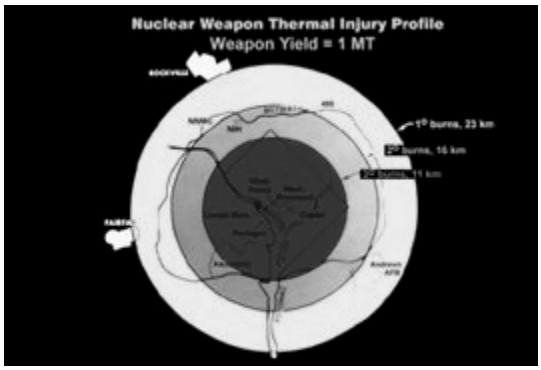

Figure 5-8. One megaton weapon thermal injury profile.

5-11. Diagnosis

Certain factors are of prime importance in the early evaluation of bums because of their relation to overall prognosis. These factors include--

- Area of the burn expressed in percentage of body surface involved.
- Involvement of critical organs; for example, head and neck, respiratory tract, genitalia, hands, and feet.
- Depth of burn; superficial (first- or second-degree), or deep (second degree) and full thickness (third degree).

a. Area of Burn. The most accurate way to estimate the amount of tissue injury following a burn is to measure the extent of the body surface burned. However, direct measurement is not generally possible or necessary, and a short cut method of estimating the

percent of the body surface involved can be very useful. The "Rule of Nines" method is a simple and reasonably reliable guide in which the various parts of the body are divided into surface areas of 9 percent each (or multiples of 9 percent) as shown in Table 5-6.

Table 5-6. Rule of Nines for Establishing Extent of Body Surface Burned

Anatomic Surface	**% of Total Surface**
Head and Neck	9 = 9
Anterior Trunk	2 x 9 = 18
Posterior Trunk	2 x 9 = 18
Upper Limbs	9 ea = 18
Lower Limbs	18 ea = 36
Genitalia and Perineum	1 = 1

(1) As the percent of the surface burned increases, morbidity and the probability of mortality increases sharply. Burns that cover 20 percent or more of the body surface can be fatal without treatment. Even with treatment, mortality from extensive burns will be high, particularly in the very young or the aged. Young healthy soldiers who have uncomplicated burns may survive even extensive involvement with proper care.

(2) Determination of the percent of the body involved will aid in planning resuscitative treatment and estimating fluid requirements during the first 48 hours after the burn injury. Patients with severe burns will suffer quite extensive fluid and electrolyte losses, resulting in severe hypovolemic shock requiring aggressive fluid replacement therapy as early as possible. An outline of a resuscitative program is given in paragraph 5-13

b. Involvement of Critical Organs. When certain organ systems are involved, the clinical effects of burns can be quite serious in spite of the fact that only a small fraction of the body is involved.

(1) *Head and neck burns.* Burns of the face can be serious problems, even if the eyes are not involved. Burns of the head frequently are complicated by severe edema, which can result in respiratory obstruction. This can be quite serious when the inhalation of hot gases has occurred. It may be necessary to do tracheotomies on many of these patients.

(2) *Burns of the respiratory tract.* When hot gases are inhaled, a very serious type of injury may be sustained. These injuries have a high probability of mortality if the burns extend deep into the alveoli. These patients are very fragile and may not tolerate early evacuation. Pulmonary edema may develop abruptly, without warning, requiring vigorous ventilator support. These injuries can be very difficult to manage.

(3) *Burns of hands and feet.* These can be very disabling and may require long hospitalization for extensive surgical care even though they are not life-threatening injuries.

These patients may not be able to care for themselves and, as a result, will require extensive nursing care.

c. *Depth of Burn.* Burns are classified on the basis of the depth of the injury.

(1) *Superficial or partial skin thickness burns.* These are lesions in which the dermis is intact and only the epidermis is injured. When the injury is limited and only erythema occurs (such as in a sunburn), these are usually called first-degree burns. If blistering is seen, the injuries are called second-degree burns. Superficial burns are usually painful but will heal readily by epithelization unless infection occurs. Infection can convert a typical second-degree, superficial burn into a deep or full-thickness burn that will not heal by epithelization but rather by scarring. Second-degree burns will be very common in nuclear combat and may be the one of the most common injuries seen.

(2) *Deep or full-thickness burns.* Injuries involving the full thickness of the skin which cannot heal by epithelization are called third-degree burns. Instead, these injuries heal by scarring, and as a result there may be contraction and loss of function, particularly if extremities are involved. Extensive plastic surgery may be required to prevent or limit loss of function. The areas of a burn that are third degree are usually painless, and this helps differentiate areas of third from second-degree when both are present. The earlier the diagnosis of the degree of burn is made, the sooner reconstructive treatment with skin grafting can be started. In general, however, in nuclear combat, early skin grafting will rarely be possible.

5-12. First Aid

a. Thermal burns caused by fire, hot objects, hot liquids, and gases or by a nuclear detonation or fireball often cause extreme pain, scarring, or even death. Proper treatment will minimize further injury of the burned area. General first aid procedures include the following:

- Perform basic lifesaving measures.

- Remove the casualty quickly and cover the *thermal burn* with any large nonsynthetic material, such as a field jacket. Roll the casualty on the ground to smother the flames.

- Cut and gently lift away any clothing covering the burned area, without pulling the clothing over the burns. Leave in place any clothing that is stuck to the burns. If the casualty's hands or wrists have been burned, remove jewelry if possible without causing further injury and place in his pockets. This prevents the necessity to cut off jewelry since swelling usually occurs as a result of a burn.

- Apply a field dressing to the burn.

- **Do not--**

- Place the dressing over the face or genital area.
- Break the blisters.
- Apply grease or ointments to the burns.
- If the casualty is conscious and not nauseated, give him small amounts of water.

b. When an individual suffers thermal burns of the face from a fire, the eyes will close quickly due to extreme heat. This reaction is a natural reflex to protect the eyeballs; however, the eyelids remain exposed and are frequently burned. If a casualty receives burns of the eyelids/face, **do not** apply a dressing; **do not touch**; and personnel should seek medical treatment immediately.

5-13. Treatment

Initial treatment of burn patients will be resuscitative. When such patients are first seen, a simple plan of treatment must include maintenance of airway with ventilating support as needed; providing adequate fluid therapy; and carefully maintaining records of input and output.

a. Maintenance of Airway. This is of particular importance in head and neck burns or in unconscious patients. If large numbers of patients are seen requiring transportation over long distances early in the postburn period, tracheotomies may have to be done on a routine basis. Tracheotomies done prior to the onset of edema are much easier to perform than when they are done after edema has resulted in respiratory obstruction. When only small numbers of patients require treatment, tracheotomies are rarely required.

b. Fluid Therapy. The shock that is associated with an extensive burn will be severe, and survival of these patients depends upon adequate, balanced fluid replacement therapy. In combat, however, standardized methods of management are required. Standard formulae for determining the fluid requirements of burn patients have been developed and can be used in combat. The basic principle in these formulae is that the amount of fluid required is proportional to the percent of body surface burned and body weight. The type of fluid used includes colloidal materials to replace the plasma constituents lost as well as electrolytes.

(1) *Fluid requirements for first 24 hours.* Fluid requirements are as follows:

- Colloid solutions: 0.5 ml x body weight in kilos x percent of body surface burned.
- Electrolyte solutions: 1.5 x body weight in kilos x percent of body surface burned.
- Additional fluids: 2,000 ml 5 to 10 percent dextran in water.

As an example, to meet the requirements of a 70-kg person with 30 percent body surface burn, the formula would be--

Colloid: 0.5 ml x 70 x 30 = 1,050 ml
Electrolyte: 1.5 ml x 70 x 30 = 3,150 ml
Metabolic Fluid (carbohydrates): 2,000 ml
Total: 6,200 ml

(2) *Restrictions.* Certain restrictions on the application of this formula are required since it is only a guide.

- Fluid requirements for an injury involving more than 50 percent of the body surface should be calculated as if the burn were no more than 50 percent.

- The maximum of fluid given in the first 24 hours should be 10,000 ml.

- The first half of the fluid should be given more rapidly than the second; and the actual rate of administration should be adjusted according to urinary output.

- During the second 24 hours, the colloid and electrolyte given should be about one-half of that given during the first 24 hours. Again, the actual rate should be adjusted to maintain a reasonable urinary output. This is the single best clinical guide to use in determining the patient's actual fluid requirements.

- After the 3rd or 4th day, the patients will begin to resorb fluid from the edematous areas and will excrete it in large quantities. Administration of fluids to replace this loss is contraindicated. Excessive administration of fluids must be avoided during this time, and fluid intake can generally be reduced to that normally required for metabolic needs.

c. Input and Output Records. It is extremely important to accurately follow the input and output of fluids in burn patients. It would be impossible to modify fluid therapy according to individual needs without accurate records. Combat medical records, however, must be simple and should be attached to the patient so that they accompany him during evacuation. Medical planners must consider how to modify and improve combat medical records so that accurate input and output data on burn patients can be recorded. Most burn patients will require urinary catheterization, and this can aid considerably in recording urinary output rates accurately.

d. Care of Burn Wound. Although the first priority in patient care is resuscitation, proper care of the burn wound is essential both for survival as well as for optimum healing and preservation of function. In that regard, as soon as the patient's overall condition permits, after hospitalization, initial debridement and cleaning of the burn should be done. The main purpose of this treatment is to remove foreign material and dead tissue to minimize infection. Thorough irrigation and the application of topical antimicrobial creams such as argentic sulfadiazine and sterile dressings should complete the initial procedures. Special attention should be given to critical areas such as the hands and surfaces over joints.

CHAPTER 6

BIOLOGICAL AND MEDICAL EFFECTS OF IONIZING RADIATION: DIAGNOSIS, TREATMENT, AND PROGNOSIS

Section I. BASIC BIOPHYSICAL ACTION AND CELLULAR EFFECTS OF IONIZING RADIATION

6-1. General

a. This section will cover basic biophysical and biological effects of ionizing radiation in order to form a foundation for understanding the clinical aspects of radiation injury covered later in this chapter. This extended discussion of radiation does not imply that nuclear radiation will be the most important cause of casualties after a nuclear explosion. Blast and thermal injuries in many cases will far outnumber radiation injuries. However, radiation effects are considerably more complex and varied than are blast or thermal effects, and are also subject to considerable misunderstanding. Since data from human experience are limited, much of the information in this section is based upon experimental information from animal studies.

b. A wide range of biological effects may follow the exposure of an animal to ionizing radiation. These may include rapid death following extremely high radiation doses of penetrating whole-body radiation, or delayed radiation effects following lower doses. The nature and severity of these effects will depend upon a variety of biological and physical factors. There are significant variations in response to a radiation exposure associated with differing biological factors such as animal species, age, as well as the physical factors of dose, dose rate, and nature of the radiation. However, the biological responses to radiation are not unique. They fall within the range of standard tissue responses seen following other types of injury and occur as a result of similar biochemical and/or cell kinetic disturbances. As a result, the wide range of effects that are possible can be organized into a predictable scheme, the details of which form the basic material in this section.

6-2. Ionizing Radiation

There are only four types of ionizing radiation associated with atmospheric and underground nuclear detonations of biological significance. These are X and gamma rays (photons), neutrons, beta particles, and alpha particles. Aspects of their mechanisms of interaction with living tissue are summarized here.

a. *Gamma and X-Ray Photon Radiation.* Gamma radiation is in the form of electromagnetic waves (photons) emitted by nuclear decay during the nuclear detonation, or later in fallout. X-rays are also electromagnetic waves (photons), but they are emitted from the electron shell due to electron energy shifts. Photons are highly penetrating and a large fraction may pass through the human body without interaction. Consequently, energy deposition can occur anywhere in the body along a given gamma photon's path. A significant portion of the

body may be exposed to gamma radiation during a nuclear detonation in combat or because of an industrial accident. This is in marked contrast to the highly localized exposure pattern that occurs with alpha and beta radiation. High-energy gamma emitters deposited within the body may also result in total body irradiation just as effectively as external sources. This may occur even when the gamma emitting nuclides are not distributed uniformly throughout the body.

b. Neutron Radiation. Neutrons are uncharged particles and react primarily with atomic nuclei along their path through the human body. The probability of interaction of neutrons in the energy range characteristic of a nuclear detonation's fission neutron spectrum is roughly comparable to that of low-energy gamma photons. The energy deposition will not be uniform, and the side of the body facing the detonation will absorb more energy than the opposite side. The uneven nature of energy deposition will result in remarkably different biological effects as compared to those of gamma and x-ray radiation.

(1) *Slow* neutrons interact with atomic nuclei directly. Because of their mass and energy, fission spectrum neutrons can disrupt atomic structure, typically causing a *recoil escape* of a target nucleus from its orbital electrons. These elastic collisions are much more common with the very light atoms, particularly hydrogen, the major constituent atom of living tissue. These recoil nuclei are capable of causing dense ionization and formation of hydroxyl radicals along their paths.

(2) In biological material, elastic collisions between neutrons and the nuclei of light atoms predominate. These recoil nuclei will expend their energy along short tracks of high excitation and ionization density. In tissue, about 70 percent to 85 percent of the entire fast neutron energy is transferred to recoil hydrogen nuclei (a single proton). The similarity of neutron and proton masses allows energy transfer much like that between billiard balls. The remainder of the neutron energy is dissipated in recoil nuclei of other atoms.

(3) After the neutrons have lost most of their energy through these collisions, they will reach a temperature equilibrium energy state and are called thermal neutrons. These thermal neutrons have a high probability of being captured by the nuclei of other elements such as sodium. A gamma photon is produced on this neutron capture reaction and the resulting atom is radioactive. *This secondary tissue radiation is not a significant factor in radiation injury*. However, if the radioactivity produced can be measured, it can be used in the estimation of neutron doses.

c. Beta Radiation.

(1) Beta rays are energetic electrons emitted from atomic nuclei and can only penetrate a few millimeters of tissue. If the beta-emitting material is on the surface of the skin, the resulting beta irradiation causes damage to the epithelial basal stratum. The lesion initially appears similar to a superficial thermal burn but significantly more damage normally has occurred. However, if the radionuclide is incorporated internally, the damage will be in small spheres of tissue around each fragment or radioactive source. The total tissue damage is a function of the number of such sources within the affected tissue volume, the nuclide's intrinsic radioactivity, and the radiosensitivity of the tissue.

(2) Dead cells are replaced quickly in most tissues. Unless the individual cells that were involved are highly critical or the fraction of cells killed in a given organ is large, individual cell death does not cause significant clinical effects. Beta radiation may simply damage rather than kill affected cells. Damaged cells may be of greater significance, particularly if they become malignant or otherwise malfunction. The less dense energy deposition of beta radiation may therefore result in late pathologies.

d. Alpha Radiation.

(1) If the source of the radiation is external, all of the alpha radiation is absorbed in the superficial layers of dead cells within the stratum corneum. Even if tissue paper is interposed, the alpha particles will be absorbed and not reach the skin. Because of this, alpha radiation is not an external hazard. If alpha-emitting material is internally deposited, all the radiation energy will be absorbed in a very small volume of tissue immediately surrounding each particle. Alpha radiation has such limited penetrating ability that the maximum range for the highest energy alpha particle in tissue is less than 0.1 mm. Thus, while extremely high radiation doses may be deposited in the few cells immediately surrounding a source of alpha radiation, regions outside this irradiated spherical volume are not affected. Beyond a radius of about 0.02 millimeters, the deposition of energy is very small. The high radiation doses within this critical radius are lethal to the cells immediately adjacent to the source. These cells are then removed by phagocytosis or undergo fibrosis. Relatively little damage to the intact organism results, unless these cells are themselves highly critical. Although internal alpha radiation can be lethal to individual cells, the overall acute injury is small. However, internal deposition of alpha particles is important in terms of causing long-term radiation injury. It will not cause acute performance degrading symptoms.

(2) Many alpha-emitting materials also emit gamma radiation, and this gamma radiation may cause significant injury, even though the total alpha energy exceeds the total gamma energy and the ratio of gamma emissions per alpha is very small. This follows from the fact that the penetrating power of gamma radiation is many times greater than that for alpha radiation so that the total volume of tissue exposed to damaging radiation is much greater.

6-3. Relative Biological Effectiveness

a. Relative biological effectiveness (RBE) is defined as the ratio of the absorbed dose of the reference radiation (250 kVp X rays) to the absorbed dose of a test radiation to produce the same level of biological effect, all other conditions being equal. When two radiations produce a biological effect that is not of the same extent and/or nature, the RBE cannot be specified.

b. Primates irradiated with neutrons simulating a nuclear detonation demonstrate marked changes in behavior, vomiting, neurological symptoms, CV disorders and other symptoms. The fission spectrum neutron RBE for these disturbances was between 0.5 and 1.2 in the dose range from 0.5 to 6.5 Gy. The importance of intermediate dose and the biological

effects as causes of incapacity are significant. In operational terms, neutron RBE varies with neutron energy, with neutron dose (the size of the neutron dose/gamma dose ratio), the dose rate and above all the dose gradient, particularly for determination of hematological LD_{50}, but doubtless also for vomiting and early transient incapacitation (ETI). The RBE for ETI has been established as being equal to 1, because insufficient evidence has been collected to indicate otherwise. Relating dose to radiation effects in man and other large mammals is further complicated by the fact that mixed-spectrum radiations change as they interact with body tissue. Gamma photons have been found more effective in producing early transient incapacitation than either high-energy neutrons or fission spectrum neutrons.

6-4. Linear Energy Transfer Quality Factor and the Radiation Weighting Factor

These concepts were discussed in Chapter 3, but are reviewed here in the context of cellular interaction.

a. As a radiation passes through an absorbing material, it will lose some of its energy through interactions with atoms and molecules along its path. An increase in the number of interactions leads to an increase in the amount of energy deposited in the absorbing medium. The LET of a radiation is defined as the average energy locally deposited by a radiation per unit length traversed in an absorbing medium. The higher the LET, the more effective the radiation is at producing biological damage. Low LET radiations are generally sparsely ionizing and have a random potential for interaction along the path traversed. These radiations can easily penetrate materials making them an external radiation hazard. Conversely, high LET radiations are densely ionizing and are more uniformly ionizing, but they penetrate materials poorly and pose a hazard only if internalized.

b. Linear energy transfer is primarily of importance in occupational/low-level dose exposures, and has little relevance to military combat operations. As each type of radiation has a unique LET, direct comparisons cannot be made among absorbed doses of different radiations. For example, one Gy of alpha radiation will not produce the same biological effect as one Gy of gamma radiation. A direct comparison of doses from different radiations can only be made after normalization to a common scale. The radiation-weighting factor, developed by the International Commission on Radiological Protection (ICRP) provides a way to do this. (See Table 6-1.)

c. The NRC calls its radiation-weighting factor the quality factor. The NRC utilizes the traditional radiation units, *rem* to express the quality factor, and calls the radiation-weighted dose the *dose equivalent*. As the NRC only has jurisdiction within the US, this document will use the *Sv* instead of the *rem*. To minimize confusion, the generic term dose, or effective dose, is commonly used when referring to the radiation-weighted dose instead of *equivalent dose* or *dose equivalent*.

Table 6-1. Quality and Radiation Weighting Factors for Various Radiation Types

Radiation	Quality	Weighting Factors
X-, gamma-, and beta-rays	1	1
Neutrons*		
Thermal	2	5
Fast	10	20
Alpha particles, fission fragments, and heavy nuclei	20	20

* Values of Quality factors and Weighting factors for neutrons are dependent upon the energy of the neutron.

6-5. Cellular Effects of Ionizing Radiation

Observed cellular effects of radiation are similar for different kinds and doses of ionizing radiation.

a. Changes in Cell Function. One of the simplest effects to observe is cell death since radiation often induces apoptosis (programmed cell death, usually by fragmentation). Non-lethal changes in cellular function can occur as a result of lower radiation doses. These include delays in certain phases of the mitotic cycle, disrupted cell growth, permeability changes, and changes in motility.

(1) *Mitotic cycle.* Mitosis may be delayed or inhibited following radiation exposure. Dose dependent inhibition of mitosis is particularly common in actively proliferating cell systems. This inhibition occurs approximately 40 minutes before prophase in the mitotic cycle, at a time when the chromosomes are discrete, but prior to the breakdown of the nuclear membrane. Subsequent irradiation after this radiation transition point does not delay mitosis. Delays in mitosis can cause profound alterations in cell kinetic patterns resulting in depletions of all populations. This is the basic mechanism underlying the later clinical changes seen in the hematopoietic and GI syndromes of whole-body irradiation.

(2) *Disruptions in cell growth.* Cell growth may also be retarded, usually after a latent period. This may be due to progressive formation of inhibitory metabolic products and/or alterations in the cell microenvironment.

(3) *Permeability changes.* Irradiated cells may show both increased and decreased permeability. Changes due to irradiation within the lipid bilayers of the membrane may alter ionic pumps. This may be due to changes in the viscosity of intracellular fluids associated with disruptions in the ratio of bound to unbound water. Such changes would result in an impairment of the ability of the cell to maintain metabolic equilibrium and could be very damaging even if the shift in equilibrium were quite small.

(4) *Changes in cell motility*. The motility of a cell may be decreased following irradiation. However, the presence of normal motility does not imply the absence of radiation injury. Irradiated spermatozoa, for example, may retain their motility and be capable of fertilization while carrying radiation-induced genetic changes, which may alter subsequent embryogenesis.

b. Relative Cellular Radiosensitivity. In general, actively proliferating cells are most sensitive to radiation. On the other hand, the mitotic activity of all cells decreases with maturation. Thus, cellular radiosensitivity tends to vary inversely with the degree of differentiation. Cells may be classified functionally, and in decreasing order of sensitivity into four categories--vegetative cells, differentiating cells, totally differentiated cells, and fixed nonreplicating cells.

(1) *Vegetative cells.* These cells, comprising differentiated functional cells of a large variety of tissues, are generally the most radiosensitive. Examples include--

- Free stem cells of hematopoietic tissue (hemocytoblasts, primitive lymphoblasts, primitive erythroblasts, and primitive myeloblasts).
- Dividing cells deep in the intestinal crypts.
- Primitive spermatogonia in the epithelium of the seminiferous tubules.
- Granulosa cells of developing and mature ovarian follicles.
- Basal germinal cells of the epidermis.
- Germinal cells of the gastric glands.
- Large- and medium-sized lymphocytes.
- Small lymphocytes, which are not included normally in this class of cells, but which are also highly radiosensitive.
- Mesenchymal cells.

(2) *Differentiating cells*. These cells are somewhat less sensitive to radiation. They are relatively short-lived and include the first generation produced by division of the vegetative mitotic cells. They usually continue to divide a limited number of times and differentiate to some degree between divisions. As differentiation occurs, radiosensitivity decreases. The best examples of this type of cell are the dividing and differentiating cells of the granulocytic and erythrocytic series in the bone marrow. This type also includes the more differentiated spermatogonia and spermatocytes in the seminiferous tubules and the oocytes.

(3) *Totally differentiated cells.* These cells are relatively radio resistant. They normally have relatively long life spans and do not undergo regular or periodic division in the adult stage, except under abnormal conditions such as following damage to, or destruction of a large number of their own kind. This class includes hepatocytes, cells of interstitial gland tissue of the gonads, smooth muscle cells, and vascular endothelial cells.

(4) *Fixed nonreplicating cell*s. These cells are the most radioresistant. They do not normally divide, and some types, such as neurons, do not divide under any circumstances. They are highly differentiated morphologically and highly specialized in function. Cells in this group have widely varied life spans and show progressive aging. This group includes the long-lived neurons, striated muscle cells, short-lived polymorphonuclear granulocytes and erythrocytes, spermatids and spermatozoa, and the superficial epithelial cells of the alimentary tract.

6-6. Relative Organ Radiosensitivity

The relative radiosensitivity of an organ depends upon its component tissue sensitivities. Table 6-2 lists various organs in decreasing order of radiosensitivity, based on a direct radiation effect, parenchymal hypoplasia. Each of the numerous cell renewal systems making up total cellular mass is normally in an equilibrium state between cell formation, proliferation, maturation, and death. Some systems, such as the adult CNS in higher animals, are stabilized at the end point of maturation, and the functional cells of such a system are not replaced if lost or destroyed. Other organ systems, such as the liver, which do not normally replace cells at a rapid rate, have the potential to regenerate large numbers of cells if needed. Other organ systems, such as the skin, the reproductive system, the GI tract, and the hematopoietic system in the bone marrow, maintain a continuous high cell turnover rate. Effects on specific organ systems are discussed in the following paragraphs:

Table 6-2. Relative Radiosensitivity of Various Organs Based on Parenchymal Hypoplasia

Organs	Relative Radio-sensitivity	Chief Mechanism of Parenchymal Hypoplasia
Lymphoid organs; bone marrow, testes and ovaries; small intestines Embryonic tissue	High	Destruction of parenchymal cells, especially the vegetative or differentiating cells
Skin; cornea and lens of eyes; gastrointestinal organs: cavity, esophagus, stomach, rectum	Fairly high	Destruction of vegetable and differentiating cells of the stratified epithelium
Growing cartilage; the vasculature; growing bones	Medium	Destruction of proliferating chondroblasts or osteoblasts; damage to the endothelium; destruction of connective tissue cells and

		chondroblasts or osteoblasts
Mature cartilage or bone; lungs; kidneys; liver; pancreas; adrenal gland; pituitary gland	Fairly low	Hypoplasia secondary damage to the fine vasculature and connective tissue elements
Muscle; brain; spinal cord	Low	Hypoplasia secondary damage to the fine vasculature and connective tissue elements, with little contribution by the direct effects on parenchymal tissues

a. Effect on Bone Marrow Cell Kinetics. As mentioned, the hematopoietic system in the bone marrow has a high turnover rate; bone marrow also has a large reserve capacity in an adult. A large fraction of it is normally nonfunctioning but has the potential to be functional if required. The bone marrow contains three cell renewal systems--the erythropoietic (red cell), the myelopoietic (white cell), and the thrombopoietic (platelet). The time cycles and cellular distribution patterns and postirradiation responses of these three systems are quite different. Studies suggest that a phuripotential stem cell gives rise to these three main cell lines in the bone marrow. Beyond this stem cell, each cell renewal system consists of a stem cell compartment for the production of erythrocytes, leukocytes (lymphocytes, granulocytes, monocytes, and so forth), or platelets, a dividing and differentiating compartment, a maturing (nondividing) compartment, and a compartment containing mature functional cells. Research studies suggest that each of these cell renewal systems operates under the influence of regulating factors, primarily at the stem cell level, through a negative feedback system initiated in large measure by the level of mature circulating cells in the peripheral blood. Normally, a steady-state condition exists between new cell production by the bone marrow and the numbers of functional cells. Morphological and functional studies have shown that each cell line, that is, erythrocyte, leukocyte, and platelet, has its own unique renewal kinetics. The time-related responses evident in each of these cell renewal systems after irradiation are integrally related to the normal cytokinetics of each cell system.

b. Gastrointestinal Kinetics.

(1) The vulnerability of the small intestine to radiation is primarily the cell renewal kinetics of the intestinal villi. The renewal system is in the crypt and villus structures where epithelial cell formation, migration and loss occur. The four cell renewal subsets are stem and proliferating cell compartment, maturation compartment, functional compartment, and the extrusion zone. Stem cells and proliferating cells move from crypts into a maturing only compartment at the neck of the crypts and base of the villi. Functionally mature epithelial cells migrate up the villus wall and are extruded at the villus tip. The overall transit time from stem cell to extrusion on the villus for man is estimated as being 7 to 8 days.

(2) Because of the high turnover rate occurring within the stem cell and the proliferating cell compartment of the crypt, marked damage occurs in this region by whole body radiation doses above the mid-lethal range. Destruction, as well as mitotic inhibition, occurs within the highly radiosensitive crypt and proliferating cell compartments within hours after high

doses. Maturing and functional epithelial cells continue to migrate up the villus wall and are extruded, although the process is slowed. Shrinkage of villi and morphological changes in mucosal cells, occur as new cell production is diminished within the crypts. Continued extrusion of epithelial cells in the absence of cell production can result in denudation of the intestinal mucosa. Concomitant injury to the microvasculature of the mucosa and submucosa in combination with epithelial cell denudation results in hemorrhage and marked fluid and electrolyte loss contributing to shock. These events normally occur within one to two weeks after irradiation. A second mechanism of injury has recently been detected at the lower range of the GI syndrome, or before major denudation occurs at higher doses of radiation. This response is a functional increase in fluid and electrolyte secretion on the epithelial cells without visible cell damage. This second mechanism may have important implications for fluid replacement therapy.

c. Microvascular Effects. The microvasculature of all tissue and organ systems is susceptible to damage by ionizing radiation exposure. The magnitude of damage elicited and the degree to which repair ensues are dependent on the amount and duration of exposure, on the extent of tissue exposed, and the quality of radiation (high LET particle versus low LET photons).

(1) The initial damage and repair responses expressed by radiation-injured endothelia lay a common foundation for both the early arising, acute effects, as well as for subsequent late arising pathologies. Exposure-induced lesions on luminal surfaces of endothelial cells appear to provide initial sites for thrombogenic foci, that not only extends endothelial damage with resulting changes in vessel wall permeability, but also activates a reparative molecular cascade in an attempt to correct the vascular defect. The major molecular players in this cascade include von Willenbrand factor (vWf; clotting factor-8) which is released from damaged endothelial cells; the selected binding of angiogenic cytokines (angiogenic and platelet-derived endothelial growth factors) which stimulate the regrowth of damaged endothelial sites; and finally, the damage-mediated release of cytokines by blood platelets and lymphocytes. These cytokines selectively stimulate proliferation of perivascular elements damaged as a result of the early occurring permeability associated edema. Within limits of exposure, this repair sequence commonly results in a restructured, fully functional vessel.

(2) The early repair of radiation-induced vascular damage is a process not without liabilities. It has been long suspected that much of the late arising pathology is directly related to the early occurring damage/repair sequences of the microvasculature. Clear examples of this putative relationship can be readily recognized in a number of the late arising pathologies. For example, in the genesis of tissue/organ system fibrosis, chronic postirradiation thrombogenic lesions act as potent angiogenic stimuli in yielding overexuberant, aberrant proliferation of perivascular elements. A still clearer example of this process is noted in the generation of late arising hemorrhagic diathesis as a direct result of malformed, dysfunctional vasculature.

d. Teratogenic Effects. Exposure to ionizing radiation can produce very severe effects on the embryo and fetus. Teratogenesis may be observed irrespective of the radiation source, be it from industrial, medical or military sources (including nuclear weapons or RDD). The effects vary depending on the gestation age, the dose, and also the dose rate. Gestation is

divided into preimplantation, organogenesis, and fetal periods. In humans, these periods correspond approximately to 0 to 9 days, 10 days to 6 weeks, and 6 weeks to term, respectively. The four main effects of ionizing radiation on the developing organism are--growth retardation, both intrauterine and after birth; severe congenital malformations including errors of metabolism; and embryonic, fetal, or neonatal death and carcinogenesis.

(1) Growth retardation is not seen after irradiation in preimplantation. In embryos exposed to radiation in early organogenesis the gravest intrauterine growth retardation are experienced, from which animals can recover later (temporary growth retardation). Irradiation in the fetal period leads to the most pronounced permanent growth retardation.

(2) The peak incidence of teratogenesis, or gross malformations, occurs when the fetus is irradiated while in organogenesis. Radiation-induced malformations of bodily structures other than the CNS are uncommon in humans, in contrast to what has been seen in animals. Radiation-induced damage to the CNS in man is first observed at the end of organogenesis (at approximately 8 weeks gestation) and extends well into the fetal period. Data on atomic bomb survivors indicate that microcephaly may result from a free-in-air dose of 0.1 to 0.19 Gy. The incidence of pronounced mental retardation as a function of dose is apparently linear without threshold at 8 to 15 weeks, with a risk coefficient of 0.4 per Gy. The incidence is about four times lower at 16 to 25 weeks. A variety of effects has been documented in animals after irradiation during fetal stages, including effects on the hematopoietic system, liver, and kidney, all occurring, however, after fairly high radiation doses. The effects on the developing gonads have been well documented and doses close to a few tenths of a Gy to one Gy invoke fertility changes in various species.

(3) Lethality from irradiation varies with stage of fetal development. The embryonic LD is lowest during early preimplantation. Neonatal death may occur following irradiation during organogenesis. The LD_{50} dose gradually approaches the adult LD_{50} in the late fetal stages.

(4) A correlation has been found between exposure to diagnostic X-rays *in utero* and the subsequent development of childhood malignancies. Some studies imply that X-ray irradiation at low doses *in utero* increases the cancer incidence by a factor of 1.5 to 2 during the first 10 to 15 years of life. Moreover, the cancer will occur earlier in life than in unirradiated individuals. Even though there is some scientific debate over the causal relationship between irradiation *in utero* and cancer, it is assumed for practical purposes that the developing organism is susceptible to radiation-induced carcinogenesis. A dose of 0.1 Gy to the embryo during the first 6 weeks after conception is often regarded as the cutoff point. The decision to terminate a pregnancy must depend on many factors in addition to the radiation dose. These include legal and moral constraints, diabetes, hypertension, maternal age, and other appropriate considerations.

6-7. Radiation-Induced Chromosome Damage

a. Chromosomes are composed of deoxyribonucleic acid (DNA), a macromolecule containing genetic information. This large, tightly coiled, double-stranded molecule is sensitive to radiation damage. Radiation effects range from complete breaks of the nucleotide chains of DNA to point mutations, which are essentially radiation-induced chemical changes in the nucleotides which may not affect the integrity of the basic structure. Intermediate effects, such as abnormal bonding between adjacent molecules and alterations in viscosity, have also been observed. After irradiation, chromosomes may appear to be *sticky* with formation of temporary or permanent interchromosomal bridges preventing normal chromosome separation during mitosis and transcription of genetic information. Unequal division of nuclear chromatin material between daughter cells may result in production of nonviable, abnormal nuclei.

b. Laboratory studies in animals indicate increased mutation rates with small doses of radiation. As radiation dose increases, mutation induction also increases. Mutations per unit dose decrease at low dose rates, however, viable mutations are still extremely rare. Most of the mutations are lethal and thus self-limiting. It must be kept in mind that radiation doses increase natural mutation rates and that the nonlethal mutations are permanent in regard to future generations.

Section II. SYSTEMIC EFFECTS OF WHOLE BODY RADIATION

6-8. General

a. Whole body irradiation is potentially the most damaging radiation and is discussed in detail in this section. However, partial body irradiation is most likely to occur in tactical scenarios where armored vehicles and improved positions are used in the course of conventional warfare. This will cause partial exposure, and/or limit the amount of radiation actually transmitted to the body. Specific organ irradiation can occur from internal deposition and retention of radioactive materials (this is discussed in Section VII). Radiation sickness occurs after exposure to a large dose of external radiation, and the mechanisms underlying the various syndromes of severe radiation sickness are discussed below. During discussion of these mechanisms, the term *median lethal dose* (LD_{50}) may be used. Medically, it has little, or no significance, but is defined here to ease understanding of key experimental data and current thought on dose severity. The LD_{50} of radiation that will kill 50 percent of exposed persons within a period of 60 days without medical intervention ($LD_{50/60}$) is approximately 4.5 Gy. Medically, other figures of interest are the dose that will kill virtually no one (LD_5) and the dose that will kill virtually everyone (LD_{95}). Approximations of those doses are within the free in air ranges of 2 to 3 Gy and 6 to 7 Gy, respectively.

b. Radiation exposure status (RES) will also be discussed frequently in this chapter; therefore, RES must be understood to enable medical personnel to better understand collective exposure limits, triage categories, and medical follow-up criteria. NATO STANAG 2083 defines exposure criteria for the commitment of troops to a radiologically contaminated area that would result in low to high level exposures to radiation (less than 50 cGy to more than 150 cGy).

One of four RES categories is assigned to a unit depending upon its cumulative dose (see Table 6-3).

Table 6-3. Nuclear Radiation Exposure Status and Degree of Risk Exposure

Radiation Exposure Status Category [a]	**Total Past Cumulative Dose [b]**	**Possible Exposure Criteria for a Single Operation That Will not Result in Exceeding the Dose Criteria for the Stated Degree of Risk [c]**
RES-0	No Exposure.	Negligible Risk: ≤50cGy Moderate Risk: ≤70cGy Emergency Risk: ≤150cGy
RES-1	More than 0, but less than or equal to 70cGy.	Negligible Risk: ≤10cGy Moderate Risk: ≤30cGy Emergency Risk: ≤110cGy
RES-2	More than 70cGy, but less than or equal to 150cGy.	Any further exposure is considered to exceed a negligible or moderate risk. Emergency Risk: ≤40cGy
RES-3	More than 150cGy.	Any further exposure will exceed the emergency risk.

[a] Radiation exposure status categories are based on previous exposure to radiation. Reclassification of units from one radiation status category to a less serious one is made by the commander, upon advice of the surgeon, after ample observation of the actual state of the health of exposed personnel.

[b] All exposures to radiation are considered total body and simply additive. No allowance is made for body recovery from radiation injury.

[c] Risk levels are graduated within each status category to provide more strigent criteria as the total radiation dose accumulated becomes more severe. The exposure criteria given for RES-1 and RES-2 units should be used only when the numerical value of a unit's total past cumulative dose is unknown. Each of the degrees of risk can be applied to radiation hazards resulting from enemy or friendly weapons, or both.

6-9. Radiation-Induced Early Transient Incapacitation

For high radiation doses in excess of 10 Gy, ETI occurs on average within 5 to 10 minutes after acute whole-body irradiation. Lowering the dose increases the median time to ETI occurrence to 12 to 15 minutes. Performance decrement has been evaluated for numerous behavioral tasks after whole body and partial body irradiation for various radiation qualities and dose rates. Several generalizations have emerged from nonhuman experimental studies.

- Early transient incapacitation is qualitatively very similar for many behavioral tasks.

- The frequency of incapacitation within a population increases as a function of radiation dose.

- Incapacitation can be elicited by both trunk-only and head-only irradiation.

- Neutrons are less effective in producing ETI than are gamma rays. For ETI, the RBE of neutron to gamma has been estimated to be between 0.23 and 0.62.

a. The frequency of incapacitation produced by a given radiation dose is proportional to the demands or the level of stress of the task being performed. Current combat casualty criteria are based on the incapacitating dose levels for both physically demanding tasks and undemanding tasks. They do not include combat ineffectiveness due to partially degraded performance that may result from slower reaction to the task, task stress, or prodromal effects of acute radiation sickness. Exposure to doses of ionizing radiation of the order of 25 Gy results in an immediate precipitous decline in cerebral blood flow (CBF) which is followed by a partial recovery at 20 to 30 minutes and subsequent slower secondary decrease in CBF thereafter, accompanied by parallel changes in systemic blood pressure. These data indicate that radiation adversely affects the ability of the brain to regulate its blood supply. The activity of certain brain enzymes involved in neurotransmitter metabolism is also considerably affected during ETI.

b. For yields of 5 to 10 KT or less, initial nuclear radiation will be the dominant casualty producer on the battlefield. Military personnel receiving an acute incapacitation dose of 30 Gy will become performance degraded almost immediately and combat ineffective within several hours. However, they will not die until 5 to 6 days after exposure if they do not receive any other injuries that make them more susceptible to the radiation dose. Military personnel receiving less than a total of 1.5 Gy will remain combat effective. Between those two extremes, military personnel receiving doses greater than 1.5 Gy will become performance degraded and some personnel will eventually die unless they receive medical intervention. A dose of 5.3. to 8.3 Gy is considered lethal but not immediately incapacitating. Personnel exposed to this amount of radiation will become performance degraded within 2 to 3 hours, depending on the physical demands of the tasks they must perform. They will remain in this degraded state at least 2 days. Adequate provision of medical care during this time frame should reduce mortality to less than 10 percent. If medical care is not provided, these personnel will experience a recovery period and be effective at performing nondemanding tasks for about 6 days, after which they will relapse into a degraded state of performance and remain so for about 4 weeks. At this time they will begin exhibiting immune incompetence radiation symptoms of sufficient severity to render them totally ineffective. Death follows at approximately 6 weeks after exposure for those individuals who do not receive appropriate evacuation and expeditious medical treatment.

c. Primates irradiated with neutrons simulating a nuclear detonation demonstrate marked changes in behavior, vomiting, neurological symptoms, CV disorders, and other symptoms. The fission spectrum neutron RBE for these disturbances was between 0.5 and 1.2 in the dose range from 0.5 to 6.5 Gy. The importance of intermediate dose and the biological

effects as causes of incapacity are significant. In operational terms, neutron RBE varies with neutron energy, with neutron dose (the size of the neutron dose/gamma dose ratio), the dose rate and above all the dose gradient, particularly for determination of hematological LD_{50}, but doubtless also for vomiting and ETI. The RBE for ETI has been established as being equal to one, because insufficient evidence has been collected to indicate otherwise. Relating dose to radiation effects in man and other large mammals is further complicated by the fact that mixed-spectrum radiations change as they interact with body tissue. Gamma photons have been found more effective in producing early transient incapacitation than either high-energy neutrons or fission spectrum neutrons.

6-10. Reproductive Cell Kinetics and Sterility

a. Despite the high degree of radiosensitivity of some stages of germ cell development, the testes and ovaries are only transiently affected by single sublethal doses of whole body irradiation and generally go on to recover normal function. In male test animals, whole body irradiation above 0.12 Gy causes abrupt decreases in sperm count. The degree of decrease is dose dependent, but a transient azoospermia will appear at sublethal radiation doses. The resulting sterility may last several months to several years, but recovery of natural fertility does occur. The recovery rate depends upon the regeneration of those elements of the stem cell population, which are in a relatively resistant part of the germ cell cycle.

b. When chromosome aberrations are produced in somatic cells, the injury is restricted to the specific tissue or cell system. However, when aberrations occur in germ cells, the effects may be reflected in subsequent generations. Most frequently, the stem cells of the germ cell line do not develop into mature sperm cells or ova, and no abnormalities are transmitted. If the abnormalities are not severe enough to prevent fertilization, the developing embryos will not be viable in most instances. Only when the chromosome damage is very slight and there is no actual loss of genetic material will the offspring be viable and abnormalities be transferable to succeeding generations. These point mutations become important at low radiation dose levels. In any population of cells, spontaneous point mutations occur naturally. Radiation increases the rate of these mutations and thus increases the abnormal genetic content of future cellular generations.

6-11. Recovery

a. Repopulation occurs by stem cell proliferation and is a particularly important recovery mechanism for both the bone marrow and the GI tract whenever the radiation exposure has been large enough to reduce cell numbers. Stem cells divide normally in both these tissues, because stem cell turnover is required to compensate for the normal continuous removal of differentiated cells. Stem cell division can be accelerated by large doses of radiation, just as any other severe insult would do. The effects of small doses are not recognized soon enough for accelerated proliferation to take place.

b. In bone marrow, large macrophage cells produce factors and cytokines that either stimulate or shut down the stem cells that are the progenitors of the erythropoietic, granulopoietic, or thrombopoietic series of blood cells. The "factor producing" cells influence one another and depress the production of one factor while the opposite is being produced. Stem cell responses continue until the factor is changed. Some stem cells have the ability to cycle at faster rates than others do but with lower efficiency, producing fewer mature cells eventually than the slower cycling cells. If the duration of exposure is sufficiently prolonged and the continued exposures are sufficiently large, then the repopulation process may become less efficient. Several months may be required before the repopulation process becomes significantly impaired and so it is not likely to be relevant in a short duration nuclear warfare scenario. This impairment may occur and contribute to the *chronic radiation syndrome* described by Russian scientists in studies of populations in heavily contaminated regions of the former Soviet Union.

Section III. RADIATION SYNDROMES

6-12. Acute Radiation Syndrome

Acute radiation syndrome is characterized by the body's polymorphic response to high dose-rate exposure to ionizing radiation. Acute radiation syndrome has been encountered after the detonation of nuclear weapons, in industrial radiation accidents, in radiotherapy patients, and in the emergency workers who responded to the Chernobyl disaster. Acute radiation syndrome has been broken down into three subsyndromes, which are dose and time profile dependent. The three syndromes described follow a similar clinical pattern that can be divided into three phases: An initial or prodromal phase occurring during the first few hours after exposure; a latent phase, which becomes shorter with increasing dose; and the manifest phase of clinical illness. The time of onset and degree of the transient incapacitation of the initial phase, the duration of the latent period, as well as the time of onset and severity of the clinical phase and ultimate outcome are all to a variable extent, dependent upon total dose and individual radiation sensitivity. Commanders and military personnel need to know the basic course of the disease and the necessity for treatment. Medical planners must be aware of the time-phased logistical requirements for treatment areas, evacuation, medical personnel, and supplies.

a. Prodromal Phase.

(1) The initial phase of prodromal symptoms (prodrome) is characterized by the relatively rapid onset of nausea, vomiting, and malaise. This is a nonspecific clinical response to acute radiation exposure. The rapidity of onset and duration of symptoms are associated with acute doses of radiation, but are not diagnostic of the degree of radiation injury. An early onset of symptoms in the absence of associated trauma does suggest a large radiation exposure. Radiogenic vomiting should not be confused with psychogenic vomiting which results from stimulation of the CNS by the sight/odor of blood, mutilation, vomitus, or excrement.

(2) The 5-HT_3 oral prophylactic antiemetics, for example, granisetron and ondansetron, will considerably diminish the reliability of nausea and emesis as indicators of

radiation exposure. Doctrinal use of these prophylactic medications may be indicated in situations where the potential for high dose radiological exposure is likely or unavoidable. These medications will diminish the nausea and vomiting in a significant percentage of those personnel exposed and consequently decrease the likelihood of a compromised individual being injured because he was temporarily debilitated. The prophylactic antiemetics do not change the degree of injury due to irradiation and are not radioprotectants in the classical sense.

b. Latent Phase. Following recovery from the prodromal phase, there will be a latent phase during which the exposed individual will be relatively symptom free. The length of this phase varies with the dose and the nature of the later clinical phase. The latent phase is longest preceding the bone-marrow depression of the hematopoietic syndrome and may vary between 2 and 6 weeks. It is somewhat shorter prior to the GI syndrome, lasting from a few days to a week. It is shortest of all preceding the neurovascular syndrome, lasting only a matter of hours. These times are exceedingly variable and may be modified by the presence of other disease or injury. Because of the extreme variability, it is not practical to hospitalize all personnel suspected of having radiation injury early in the latent phase unless radiation injury has reliably been diagnosed. Instead, it is much more reasonable to wait until the onset of the phase of clinical illness or the development of significant hematopoietic suppression as indicated by the individual peripheral blood profile.

c. Manifest Illness Phase. This phase presents the clinical symptoms associated with the major organ system injured (marrow, intestine, neurovascular system). A summary of the medical aspects of acute radiation injury and the doses at which they would be seen in young healthy adults exposed to short, high dose single exposures is shown in Tables 6-9 through 6-12. The details of the clinical courses of each of the three syndromes are described below.

6-13. Acute Radiation Component Syndromes

Acute radiation syndrome is a direct result of exposure to doses of ionizing radiation above 0.7 Gy. Its subsyndromes are dose dependent and interrelated. As described above, the higher the dose, the sooner the onset of symptoms. Consequently, casualties who acutely develop the extreme high-dose neurological illness will undoubtedly succumb to it prior to their development of the lower dose hematopoietic signs. The primary cause of death from ARS is sepsis due to hematopoietic failure. Those individuals who die with the GI syndrome usually succumb to sepsis. The neurovascular syndrome is rapidly fatal due to acute microvascular changes and membrane permeability. Survival of hematopoietic syndrome will be based on early diagnosis and institution of marrow resuscitative efforts. Interventional therapy should be begun as soon as the diagnosis of severe radiation injury is made. The archaic view that radiation injury is untreatable will be responsible for unnecessary death and debility. Medical planners and providers must ensure that prompt and appropriate care is available to personnel who receive radiation injury.

a. Hematopoietic Syndrome. Patients who have received doses of radiation in the potentially low to mid-lethal range (2 to 6 Gy) will have depression of bone marrow function with cessation of blood cell production leading to pancytopenia. Changes within the peripheral

blood profile will occur as early as 24 hours after irradiation. The exact time sequence of the depression of various circulating cell lines will vary. Lymphocytes will be depressed most rapidly and erythrocytes least rapidly. Other leukocytes and thrombocytes will be depressed somewhat less rapidly than lymphocytes. If the exposures leading to the bone marrow depression are multiple, the time of onset of depression will be very difficult to estimate. The concomitant clinical problems of a tendency toward uncontrolled hemorrhage, decreased resistance to infection, and anemia will likewise vary considerably from as early as 10 days to as much as 6 to 8 weeks after exposure. A reasonable average time of onset of clinical problems of bleeding and anemia and decreased resistance to infection is 2 to 3 weeks.

(1) *Peripheral blood count*. The most useful laboratory procedure to evaluate marrow depression is the peripheral blood count. A pancytopenia with particularly severe depression of lymphocytes, granulocytes, and thrombocyte will be strongly indicative of radiation induced bone marrow depression. Bone marrow studies will rarely be possible under field conditions and will add little information to that which can be obtained from a careful peripheral blood count. Patients will show increased evidence of hemorrhagic disease and increased susceptibility to infection. If an infection occurs, there may be little clinical response because of the concomitantly depressed inflammatory response. Death occurs from overwhelming infection and hemorrhage unless sufficient regeneration of the marrow occurs.

(2) *Erythropoiesis.*

(a) The erythropoietic system is responsible for the production of mature erythrocytes (red cells). This system has a marked propensity for regeneration following irradiation. After sublethal exposures, marrow erythropoiesis normally recovers slightly earlier than granulopoiesis and thrombopoiesis and occasionally overshoots the baseline level before levels at or near normal are reached. Reticulocytosis is occasionally evident in peripheral blood smears during the early intense regenerative phase occurring after maximum depression and often closely follows the temporal pattern of marrow erythropoietic recovery. Although anemia may be evident in the later stages of the bone marrow syndrome, it should not be considered a survival limiting sequelae.

(b) The transit time from the stem cell stage in the bone marrow to the mature red cell ranges from 4 to 7 days, after which the life span of the red cell is approximately 120 days. The immature erythroblasts and proerythroblasts undergo mitoses as they progress through the dividing and differentiating compartments. Because of their rapid proliferation, they are markedly sensitive to cell killing by ionizing radiation. Cells within the maturing (non-dividing) and functional compartments, that is, normoblasts, reticulocytes, and erythrocytes, are not significantly affected by mid-lethal to lethal range doses. The deaths of stem cells and of those within the next compartment are responsible for the depression of erythropoietic marrow. If sufficiently severe, this depression and hemorrhage are responsible for subsequent radiation-induced anemia. Because of the relatively slow turnover rate, approximately 1 percent loss of red cell mass per day, evidence of anemia is usually manifested subsequent to the depression of the other cell lines.

(3) *Lymphopoiesis.*

(a) Lymphocytes are the most radiosensitive cells of the hematopoietic system. Shortly after exposure to ionizing radiation, mature lymphocytes show early necrosis and immature splenic lymphocytes have evidence of chromatin clumping and early necrotic changes. Lymph nodes demonstrate nuclear debris within hours of irradiation. Post-irradiation cell death appears to occur in metaphase. Quantification of cells in the hematopoietic organs does not show a relationship to dose, but further cell reproduction seems to be inhibited. Surviving lymphocytes may have either an increased cellular metabolism or altered behavior.

(b) Lymphocytes disappear from the peripheral circulation in direct proportion to the dose received. The greater the radiation exposure, the more profound the lymphopenia. The leukopenia will begin within hours and proceed to its nadir within 48 to 72 hours. Experimental models have demonstrated an increase in lymph node weight over baseline at 6 months postirradiation. Atomic bomb survivors show an increased number of circulating lymphocytes.

(c) The fall in circulating lymphocytes can be utilized as a crude biodosimetry tool to estimate the effective radiation dose received. The steeper the curve, the higher the dosage and more severe the injury. For example, if a casualty's lymphocyte count drops to 25 percent of baseline at 24 hours, then a severe radiation injury has occurred and marrow resuscitative measures should be instituted as soon as is practical.

(4) *Leukopoiesis.*

(a) The function of the myelopoietic cell renewal system is mainly to produce mature granulocytes (neutrophils, eosinophils, and basophils) for the circulating blood. The most important cell type in this cell line are the neutrophils because of their role in combating infection. The stem cells and those developing stages within the dividing and differentiating compartment are the most radiosensitive. These include the myeloblast, progranulocyte, and myelocyte stages. As with the erythropoietic system, cell stages within the maturing (nondividing) compartment and the mature functional compartment (granulocytes) are not significantly affected by radiation doses within the mid-lethal range. Three to seven days are normally required for the mature circulating neutrophil to form from its stem cell precursor stage in the bone marrow.

(b) Mature functional granulocytes are available upon demand from venous, splenic, and bone marrow pools. Following an initial increase in circulating granulocytes (of unknown etiology), these pools are normally depleted before granulocytopenia is evident soon after radiation-induced bone marrow injury. Because of the rapid turnover in the granulocyte cell renewal system (approximately 8-day cellular life cycle), evidence of radiation damage to marrow myelopoiesis occurs in the peripheral blood within 2 to 4 days after whole-body irradiation. The brief latent period between the time of irradiation and the beginning depletion of circulating granulocytes is related to the transit time of the nonradiosensitive cells within the nondividing, maturing marrow compartment (metamyelocyte and band forms), during their development into mature circulating granulocytes. Recovery of myelopoiesis lags slightly behind erythropoiesis and is accompanied by rapid increases in numbers of differentiating and

dividing forms in the marrow. Prompt recovery is occasionally manifested and is indicated by increased numbers of band cells in the peripheral blood.

(5) *Thrombopoiesis.*

(a) The thrombopoietic cell renewal system is responsible for the production of platelets (thrombocytes). Platelets are produced by megakaryocytes in the bone marrow. Both platelets and mature megakaryocytes are relatively radioresistant, however the stem cells and immature stages are very radiosensitive. During their developmental progression through the bone marrow, megakaryocytic precursor cells undergo nuclear division without cell division. The transit time through the megakaryocyte proliferating compartment in man ranges from 4 to 10 days. Platelets have a life span of 8 to 9 days.

(b) As with the erythropoietic and myelopoietic systems, the time of beginning platelet depression is influenced by the normal turnover kinetics of cells within the maturing and functional compartments. Thrombocytopenia is reached by 3 to 4 weeks after mid-lethal range doses and occurs from the killing of stem cells and immature megakaryocyte stages with subsequent maturational depletion of functional megakaryocytes.

(c) Regeneration of thrombocytopoiesis after sublethal irradiation normally lags behind both erythropoiesis and myelopoiesis. Supranormal platelet numbers overshooting the preirradiation level have occurred during the intense regenerative phase in human nuclear accident victims. The mechanism of the prompt rapid recovery of platelet numbers after acute sublethal irradiation may be explained by the response of the surviving and regenerating stem cell pool to a feedback stimulus of the acute thrombocytopenic condition. Accelerated differentiation and maturation of immature megakaryocytes, as well as marked increases in size of megakaryocytes, contribute to the intense platelet production and eventual restoration of steady state levels. Blood coagulation defects with concomitant hemorrhage constitute important clinical sequelae during the thrombocytopenic phase of bone marrow and GI syndromes.

b. Gastrointestinal Syndrome.

(1) The gamma ray doses of radiation that will result in the GI syndrome are higher than those that will cause the hematopoietic syndrome alone. An acute dose that will cause this syndrome would be at least 8 Gy measured in air. Under certain circumstances, lower doses may cause this syndrome, and conversely, exposures to high doses at low dose rates or as fractionated exposures (multiple individual exposures totaling a specific dose) may not cause it. Regardless of the dose involved, the GI syndrome has a very serious prognosis, because it will almost always be accompanied by bone marrow suppression.

(2) The onset of the clinical phase of the GI syndrome occurs earlier than that of the hematopoietic syndrome. After a short latent period of a few days to a week or so, the characteristic severe fluid losses, hemorrhage, and diarrhea begin. The pathologic basis for this syndrome is an early physiologic derangement of the epithelial cells followed by a combination of severe loss of intestinal mucosa and injury to the fine vasculature of the submucosa. There is

no specific clinical sign that is pathognomonic of radiation caused GI damage. However, a peripheral blood count done on these patients should show an early onset of a severe pancytopenia occurring as a result of the bone marrow depression.

(3) In some patients, diagnosis may be difficult since sublethal hematopoietic depression (with its typical symptoms) due to radiation may, in fact, be due to some other cause such as infection. That is, it would be difficult to differentiate patients with lethal radiation sickness from those with potentially nonlethal radiation sickness complicated by dysentery. Microscopic examination of the diarrhea may reveal inflammatory cells, which is suggestive of dysentery. Radiation enteropathy is not likely to result in an inflammatory response. It must be assumed during the care of all patients that even those with a typical GI syndrome may be salvageable, until blood counts indicate that the bone marrow depression is irreversible.

c. Cardiovascular/Central Nervous System Syndrome. This syndrome is associated only with very high acute doses of radiation. The lower limit is probably 20 to 40 Gy, although hypotension (significant decline in systemic blood pressure) may be seen at even lower doses. Because of the very high doses of radiation required to cause this syndrome, personnel close enough to a nuclear explosion to receive such high doses would generally be located well within the range of 100 percent lethality due to blast and thermal effects. However, in nuclear detonations above the atmosphere with essentially no blast, very high fluxes of ionizing radiation may extend out far enough to result in high radiation doses to aircraft crews. Such personnel could conceivably manifest this syndrome, uncomplicated by blast or thermal injury. Also, personnel protected from blast and thermal effects in shielded areas could also sustain such doses. Doses in this range could also result from military operations in a reactor facility or fuel reprocessing plant where personnel are accidentally or deliberately exposed during an event where nuclear material goes critical. Very few patients will be hospitalized with this syndrome. Radiation doses in this range are uniformly fatal regardless of therapies attempted; therefore, aggressive medical support with pressors, fluids, steroids, and the like will bring only temporary improvement and may only serve to prolong suffering. Thus, therapy should be palliative in nature, and the clinician caring for such a patient is encouraged to be generous with any palliative measure such as opiates or tranquilizers.

(1) Acute radiation doses of 30 Gy and above uniformly bring death within 72 hours and usually between 24 to 48 hours, well before the insult to the GI or bone marrow systems becomes clinically apparent. Doses in this range cause significant direct effects as well as free radical overload of the cells and basement membranes of the microcirculation system. This leads to, among other damage, massive loss of serum and electrolytes through leakage into the extravascular space, circulatory collapse, edema, increased intracranial pressure, and cerebral anoxia.

(2) In less than an hour and possibly within minutes of exposure, patients receiving these doses begin experiencing prodromal symptoms: A burning sensation within minutes and severe nausea and usually projectile vomiting within an hour. The symptoms, which are severe and may last more than 24 hours, also include diarrhea that is occasionally bloody, cutaneous edema and erythema, hypotension, hyperpyrexia, disorientation, prostration, loss of coordination, and possibly seizures. Following the prodromal phase, there may be a brief

latent phase of apparent clinical improvement; but this will last only for hours to days. Finally, the victim will succumb to a complex of gross CNS dysfunction and total CV collapse, leading to a relatively prompt and inevitable death.

6-14. Pulmonary Syndrome

a. Pulmonary radiation effects will probably play a major determining role for those patients who suffer a fatal outcome from ARS. Thoracic irradiation with gamma or X-rays may cause direct radiation damage to the lung, leading to fever, coughing, chest pain, and dyspnea. Opacities will be observed in the chest radiograph corresponding to the radiation exposure field. This reaction is generally termed radiation pneumonitis. These signs and symptoms are frequently observed 2 to 10 weeks after exposure. The enormous progress made in therapeutic regimens and survivability of the hematopoietic syndrome has substantially decreased morbidity and mortality. Patients who previously might have succumbed to neutropenic-induced sepsis may well survive, but the pulmonary sequelae of their injuries have not yet been remedied.

b. Radiation pneumonitis was first described in 1923, in patients who had been irradiated for breast cancer. The minimum fractionated irradiation depth dose required to induce radiation pneumonitis in radiotherapy patients is as high as 20 to 30 Gy, while 50 to 60 Gy will almost invariably induce the disease within 5 to 6 weeks. A single exposure in the order of 8 to 10 Gy, minimum depth dose, will probably cause pneumonitis. Individual variability is large and induction may depend especially on the dose imparted to the hematopoietic system and on dose distribution. The only pulmonary first-line immune defense mechanism is formed by the alveolar macrophages. These disappear after irradiation and will not re-migrate into the lung until 4 to 6 weeks postexposure. Opportunistic microbes may grow on the denuded epithelium causing lymphocytic response, which in the chest X-ray appears as diffuse shadowing. Of 60 individuals who received an accidental acute whole body exposure of 6 to 20 Gy, 50 percent developed pulmonary signs and symptoms. Only one of these patients with radiation pneumonitis survived the first year postexposure. Those who did not develop pulmonary symptoms had a 50 percent survival.

c. When radiation pneumonitis develops and progresses over time, pulmonary fibrosis usually appears within 6 months to 1 year after exposure. Diffuse reticular and reticulonodular shadows appear in the lung fields and the lung shrinks considerably. Three types of histological lesions can be differentiated--an alveolar infiltration, an interstitial infiltration, and a mixed type. As yet, comparatively few analyses have been performed in order to actually identify the exact cause for these phenomena, although both vascular and autoallergic processes are surmised.

6-15. Cutaneous Radiation Syndrome

Acute skin injury occurs with extremely high radiation doses. Delayed, irreversible changes of the skin usually do not develop as a result of sublethal whole body irradiation, but instead follow higher doses limited to the skin. These changes are a common complication in radiation therapy,

but they should be rare in nuclear combat. They could occur with a RDD if there is heavy contamination of bare skin with beta emitter materials.

a. Cutaneous radiation effects follow a distinct clinical pattern that defines the cutaneous radiation syndrome (CRS). The different steps of development, including the symptoms, are summarized in Table 6-4. Within minutes to hours after exposure an erythematous reaction develops that may be associated with a *burning* urticaria. This transient prodromal phase usually lasts less than 36 hours. It is followed by a clinically inapparent latent phase. The manifest phase is characterized by occurrence of an intensively erythematous skin, which may show scaling and desquamation. In more severe conditions, subepidermal blisters and even ulcerations may develop. Though similar skin lesions are produced by thermal injury, the time course and underlying processes involved in the development of the CRS are so different from thermal burns that the term *radiation burns* or *beta-burns* are considered inappropriate and misleading for this clinical condition and should therefore be abandoned.

Table 6-4. Clinical Stages of the Cutaneous Radiation Syndrome

Stage	Definition	Symptoms	Occurrence	Duration	Synonyms
1	Prodromal	Erythema itch	Min- hours P.R.	4- 36 hrs	Early erythema
2a	Manifestational	Erythema	Days- 2 weeks P.R.	2- 12 weeks	Main erythema
2b		Blisters		Dry/moist desquamation *burn*	
2c		Ulcers			
3	Subacute	Erythema Ulceration	6- 9 weeks P.R.	2- 4 months	Late erythema
4	Chronic Fibrosis	6 months - Keratosis Ulceration Telangiectasias	indefinite 2 years P.R.	progressive	
5	Late	Neoplasia Ulceration Angiomas	years-decades P.R.	indefinite	

(P.R. = postradiation exposure)

b. *Chronic Cutaneous Radiation Syndrome.* In the chronic stage of the CRS, three clinical manifestations dominate the course:

- Radiation keratoses can develop in any exposed area. These lesions must be considered precancerous and should be monitored thoroughly. Single lesions may be excised.

- Radiation fibrosis is caused by an increase of collagenous tissue from dermal and subcutaneous fibroblasts and may lead to pseudoatrophy of fatty tissue. Fibrosis may lead to vasculature occlusion and cause secondary ulceration.

- Telangiectasias are a characteristic sign of the chronic stage of the CRS in humans. Apart from cosmetic disfiguring, they may cause a permanent itching sensation and a disturbing feeling of warmth.

6-16. Acute Local Radiation Injury

Local irradiation of tissues occurs when highly radioactive material, such as an industrial radiography source, is placed in proximity to tissue. As radiation intensity decreases in proportion to the square of the distance, the tissue immediately adjacent to the source receives a tremendous dosage. The total body dosage may be only 2 Gy, but the local skin dose can easily be in the hundreds of Gy (see Table 6-5).

Table 6-5. Local Tissue Damage

Dosage (Gy)	Sign	Time Post-Exposure
> 3	Epilation	2-3 Weeks
~ 6	Erythema	Minutes to Weeks
> 6	Edema	Minutes to Weeks
10-20	Blistering	2-3 Weeks
~ 30	Ulceration	1-2 Months
50-60	Gangrene, Necrosis Deep Ulceration	Weeks

a. Initial skin changes will be similar to those of CRS, but with penetrating gamma radiation, deeper tissues will express their damage over time. Development of deep-base ulcers with marked erythema at the margins is common. Granulation tissue develops poorly and months will be required for healing. Gradual expansion of the surface ulcer will occur, as the lessor damaged tissues will ulcerate later. Skin flaps will often ulcerate at the margins of attachment.

b. Deep tissues respond in a similar fashion if the source is placed in their immediate proximity. Radiotherapy literature is the best source of information concerning injury to specific tissues and anatomic structures. Table 6-6 shows the dosage received at various tissue levels after a maintenance person carried an Iridium-192 source in his pocket for 45 minutes. His whole body dose was calculated to be between 0.75 and 1.0 Gy. Acute clinical symptoms were congruent with this dose level.

Table 6-6. Acute Local Tissue Dose

Tissue Dose, 28 Ci Iridium-192 Source		
Exposure Rate: 22.5 Gy/min at 1 cm	Exposure Time: 45 min. **Radial Distance from Source**	
Tissue Depth	**0 cm**	**1 cm**
1 cm	520 Gy	160 Gy
2 cm	190 Gy	90 Gy
4 cm	52 Gy	34 Gy
8 cm	18 Gy	13 Gy

Section IV. TREATMENT OF RADIATION SYNDROMES

6-17. Diagnosis

a. The diagnosis of radiation sickness is based primarily upon the clinical picture presented by the patient. A precise history of exposure may be very difficult to obtain, since many individuals may not know that they actually have been exposed to radiation, particularly if the exposure is due to fallout, or due to exposure to a low-level radiation source. The physical findings and characteristics of the various forms of radiation sickness are described below, along with such laboratory findings as may occur. Dosimetry, at the present time, will not give an entirely adequate picture that can be used to determine either the extent of radiation injury or the prognosis. Dosimeters cannot tell whether a radiation exposure is whole body or partial body, and, they do not display the dose rate of the exposure. Finally, they cannot differentiate between single exposures and multiple exposures unless they are read at regular intervals. These unknowns, coupled with the marked effects of age or physical condition, of concomitant disease, and of combat stress, make it essential that physicians base their treatment decisions primarily upon the actual clinical condition of the patient. However, in a mass casualty situation in an operational theater, decisions based only on dosimetric data may be all that is practicable.

b. Consequently, the following guidelines apply to medical personnel operating in austere field conditions. Lymphocyte levels may be used as a biologic dosimeter to confirm the presence of pure radiation injury, but not in combined injuries. In the event of combined injuries, the use of lymphocytes may be unreliable because patients who have received severe burns or multi-system trauma often develop lymphopenia. If the physician has the resources of a clinical laboratory, additional information can be obtained to support the original working diagnosis by the presence of prodromal symptoms. An initial blood sample for concentrations of circulating lymphocytes should be obtained as soon as possible from any patient classified as *Radiation Injury Possible* or *Radiation Injury Probable*. After the initial assessment or at least no later than 24 hours after the event in question, additional blood samples should be taken for comparison. The samples may be interpreted as follows (see Figure 6-1):

- Lymphocyte levels in excess of 1500/mm^3 (cubic millimeters). The patient most likely has not received a significant dose that would require treatment.

- Lymphocyte levels between 1000 and 1500/mm^3. The patient may require treatment for moderate depression in granulocytes and platelets within 3-weeks postexposure.

- Lymphocyte levels between 500 and 1000/mm^3. The patient will require treatment for severe radiation injury. The patient should be hospitalized to minimize the complications from hemorrhage and infection that will arise within 2- to 3-weeks postexposure.

- Lymphocyte levels of less than 500/mm^3. The patient has received a radiation dose that may prove fatal. The patient needs to be hospitalized for the inevitable pancytopenic complications.

- Lymphocytes not detectable. The patient has received a superlethal radiation dose and survival is very unlikely. Most of these patients have received severe injuries to their GI and CV systems and will not survive for more than 2 weeks.

- Other guidelines. A useful rule of thumb is that if lymphocytes have decreased by 50 percent and are less than 1000/mm^3, the patient has received significant radiation exposure.

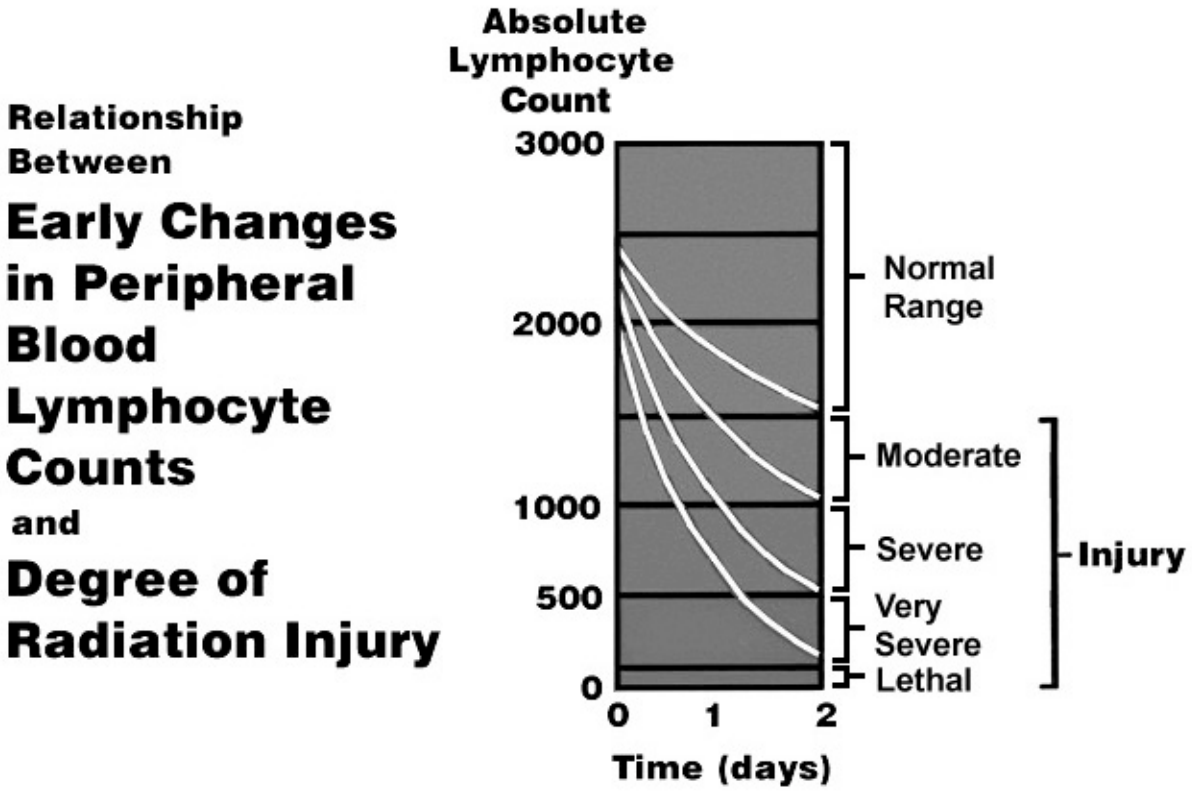

Figure 6-1. Lymphocyte nomogram.

6-18. First Aid

There is no direct first aid for radiological casualties. The first action in dealing with these casualties is to administer first aid for any conventional injuries. That is, perform the lifesaving steps, and then provide first aid for the combined injuries as described in Chapter 5.

6-19. Triage of Radiological Casualties

All combined injury patients should be treated initially as if no significant radiation injury is present. Triage and care of any life-threatening injuries should be rendered without regard for the probability of radiation or contamination. The physician should make a preliminary diagnosis of radiation injury only for those patients for whom radiation is the sole source of the problem. This is based on the appearance of nausea, vomiting, diarrhea, hypothermia, hypotension, and neurologic function. Therefore, it is best to function within a simplified, tentative classification system based on the three possible categories of patients as noted in Table 6-7 and discussed below.

a. Radiation Injury Unlikely. If there are no symptoms associated with radiation injury, patients are judged to be at minimal risk for radiation complications. These patients should be triaged according to the severity of the conventional injuries. If the patients are free of conventional injuries or diseases that require treatment, they should be released and returned to duty.

b. Radiation Injury Probable. Anorexia, nausea, and vomiting are the primary prodromal symptoms associated with radiation injury. Priority for further evaluation will be assigned after all life-threatening injuries have been stabilized. Casualties in this category will not require any medical treatment within the first few days for their radiation injuries. Evidence to support the diagnosis of significant radiation injury in the absence of burns and trauma may be obtained from lymphocyte assays taken over the next 2 days. If the evidence indicates that a significant radiation injury was received, these casualties need to be monitored for pancytopenic complications.

c. Radiation Injury Severe. These casualties are judged to have received a radiation dose that is potentially fatal. Nausea and vomiting will be almost universal for persons in this group. The prodromal phase may also include prompt explosive bloody diarrhea, significant hypotension, and signs of necrologic injury. These patients should be sorted according to the availability of resources and should receive symptomatic care. Lymphocyte analysis is necessary to support this classification.

Table 6-7. Preliminary Triage of Casualties with Possible Radiation Injuries

Possible Category of Radiation Injury			
Symptoms	**Unlikely**	**Probable**	**Severe**
Nausea	Absent	Excessive	Very Excessive

Vomiting	Absent	Present	Very Excessive
Diarrhea	Absent	Absent to Present	Absent to very Excessive
Hyperthermia	Absent	Absent to Present	Present to Very Excessive
Hypothermia	Absent	Absent	Present to Very Excessive
Erythema	Absent	Absent	Absent to Excessive
CNS Dysfunction	Absent	Absent	Absent to Excessive

d. The following symptoms frequently occur in whole body irradiated casualties within the first few hours of postexposure:

(1) *Nausea and vomiting.* Nausea and vomiting occur with increasing frequency as the radiation exceeds 1 to 2 Gy. Their onset may be as long as 6 to 12 hours postexposure, but usually subside within the first day. The occurrence of vomiting within the first two hours is usually associated with a severe radiation dose. Vomiting within the first hour, especially if accompanied by explosive diarrhea, is associated with doses that frequently prove fatal. Due to the transient nature of these symptoms, it is possible that the patient will have already passed through the initial phase of GI distress before being seen by a physician. It will be necessary to inquire about these symptoms at the initial examination.

(2) *Hyperthermia.* Casualties who have received a potentially lethal radiation injury show a significant rise in body temperature within the first few hours postexposure. Although the number of cases is few and is frequently overlooked, this condition appears to be a consistent finding. The occurrence of fever and chills within the first day postexposure is associated with a severe and a life-threatening radiation dose. Hyperthermia may occur in patients who receive lower but still serious radiation doses (2 Gy or more). Individuals wearing a chemical ensemble will normally be hyperthermic; consequently, this will not be a useful sign.

(3) *Erythema.* A person who has received a whole body dose of more than 10 to 20 Gy will develop erythema within the first day postexposure. This is also true for those who received comparable doses to local body regions, when the erythema is restricted to the affected area. Erythema is less frequently seen with lower doses (2 Gy or more).

(4) *Hypotension.* A noticeable and sometimes clinically significant decline in systemic blood pressure has been recorded in victims who have received a supralethal whole body radiation dose. A severe hypotensive episode was recorded in one person who had received several thousand rads. In persons who received several hundred rads, a drop in systemic blood pressure of more than 10 percent has been noted. Severe hypotension after irradiation is associated with a poor prognosis.

(5) *Neurologic dysfunction.* Experience indicates that almost all persons who demonstrate obvious signs of damage to the CNS within the first hour postexposure have received a superlethal dose. Symptoms include mental confusion, convulsions, and coma.

Intractable hypotension will probably accompany these symptoms. Despite vascular support, these patients succumb within 48 hours.

e. Casualties who have received a potentially fatal dose of radiation will most likely experience a pattern of prodromal symptoms that is associated with the radiation exposure itself. Unfortunately, these symptoms are nonspecific and may be seen with other forms of illness or injury, which may complicate the process of diagnosis. Therefore, the triage officer must determine the symptoms that have occurred within the first day postexposure, evaluate the possibility that they are indeed related to radiation exposure, and then assign the patient to one of the three categories--*Radiation Injury Unlikely*; *Radiation Injury Probable*; or *Radiation Injury Severe*. In the last two categories, the study of changes in circulating lymphocytes may either support or rule out the original working diagnosis. Table 6-8 summarizes the final triage classifications based upon the starting classification and estimated dose received.

Table 6-8. Final Triage Classifications

Starting Triage Classifications	**Final Triage Classifications**		
	Less Than 150 cGy	**150-550 cGy**	**Over 550 cGy**
Radiation Only	Minimal or Duty	Delayed **	Delayed
Immediate	Immediate	Immediate or Expectant *	Expectant
Delayed	Delayed	Delayed or Expectant*	Expectant
Minimal	Minimal	Minimal *	Expectant
Expectant	Expectant	Expectant	Expectant

* Select *expectant* in the case of full or partial thickness burns covering more than 18 percent of the body surface, or trauma which would either result in significant infection or be categorized as *severe* but not immediately life-threatening , such as a fractured femur. This is a clinical decision and not necessarily subjectively reproducible.
** Includes the probable requirements for antibiotics and transfusion at a later time. So this classification does not suggest that the patient is not in need of treatment, but rather that he does not need immediate specialized care.

6-20. Biodosimetry and Laboratory Testing

Detailed laboratory testing is primarily focused on protecting the patient and determining biological dosimetry. It is generally not feasible in an austere operational field environment, but would normally be possible in the aftermath of a low-level radiation incident or nuclear accident (see Appendix A). Table 6-9 lists clinical laboratory studies and the medical treatment level where they should be performed.

Table 6-9. Medical Assay of the Radiological Patient

Location / Test	Decontam-ination Point	Medical Treatment Unit (Level 2)	Hospital (Level 3)	Tertiary Care (Level 4)
Nasal Swabs for Inhalation of Contaminants	+			
External Contamination	+		+	
Urine and Stool Sample for Internal Contamination		Baseline sample	24-hr sample	+
Complete Blood Count (CBC) / platelets	If practical	Baseline sample and daily	Daily x 2 wk	Daily x 2 wk
Absolute Lymphocyte Count		Every 4-12 hrs	Every 4-12 hrs x 3 days	
Human Leukocyte Antigen (HLA) Subtyping		Draw sample	Draw sample before lymphocyte count falls	Draw sample before lymphocyte count falls
Cytomegalovirus (CMV)			+	+
Hemoglobin Agglutinin			+	+
Human Syncytial Cell Virus Antibodies				+
Human Immunovirus			+	+
Vesiculovirus				+
Lymphocyte Cytogenetics		Draw sample and send to lab	Draw sample before lymphocyte count falls	+

a. Biological dosimetry by chromosomal aberration analysis is described in Appendix A. When possible, but only after 24 hours, laboratory testing should include lymphocyte cytogenetics. Dose assessment cytogenetic peripheral blood specimens should be drawn as early as possible and transported to the closest echelon laboratory with this capability. If possible, clothing should be stored for later dosimetry evaluation.

b. In potentially lethal cases, bone marrow biopsy and aspirate should be obtained from both pelvis and sternum. Cells/sections should be frozen for future analysis. Optional testing would include thyroxin (T4), thyroid stimulating hormone (TSH) assay, serum and plasma for frozen storage, and dental biopsy for dosimetry testing. Autopsies should be performed in the case of fatal accidents, complete with photographs of relevant findings, and storage of frozen tissues.

c. No data are currently available concerning the advisability of whole body magnetic resonance imaging (MRI) in order to evaluate surviving bone marrow distribution and *in vitro* culture of bone marrow cells for hematopoietic progenitor cell growth. These studies would be indicated only in uncomplicated cases with full informed consent.

6-21. Management of the Hematopoietic Syndrome Patient

The primary goal of hematopoietic patient support is a reduction in both the depth and duration of leukopenia.

a. Conventional Therapy of Neutropenia and Infection. The prevention and management of infection is the mainstay of therapy. There is a quantitative relationship between the degree of neutropenia and the increased risk of infectious complications. Antibiotic prophylaxis should only be considered in afebrile patients at the highest risk for infection. These patients have profound neutropenia ($<0.1 \times 10^9$ cells/l or 100 cells/µl) that has an expected duration of greater than 7 days. Although the degree of neutropenia (absolute neutrophil count, ANC < 100/l) is the greatest risk factor for developing infection, other factors also influence the treatment choice and outcome. Such factors include duration of neutropenia, bactericidal functionality of surviving neutrophils, alteration of physical defense barriers, the patient's endogenous microflora, and organisms endemic to the hospital and community. As the duration of neutropenia increases, the risk of secondary infections such as invasive mycoses also increases. It is for these reasons that adjuvant therapies such as the cytokines sargramostim (GM-CSF) or filgrastim (G-CSF) will prove invaluable in the treatment of the severely irradiated person. (See Appendix C.)

b. Prevention of Infection. Initial care of medical casualties with moderate and severe radiation exposure should probably include early institution of measures to reduce pathogen acquisition, with emphasis on low microbial content food, acceptable water supplies, frequent handwashing (or wearing of gloves), and air filtration. Prophylactic use of selective gut decontamination with antibiotics that suppress aerobes but preserve ordinarily commensal anaerobes is recommended. These measures help control the alimentary canal source (mouth, esophagus, and intestines) of postinjury infections. Maintenance of gastric acidity (avoidance of antacids and H_2 blockers) may prevent bacteria from colonizing and invading the gastric mucosa and may reduce the frequency of nosocomial pneumonia due to aspiration of these organisms. The use of sucralfate or prostaglandin analogues may prevent gastric hemorrhage without decreasing gastric activity. When possible, an early oral immunocompetent diet is preferred to intravenous feeding in order to maintain the immunologic and physiologic integrity of the gut. Surgical implantation of a subcutaneously tunneled central venous catheter can be considered to

allow frequent venous access, but meticulous attention to proper care is necessary to reduce disastrous catheter associated infections.

c. Management of Infection.

(1) The management of established or suspected infection (neutropenia and fever) in irradiated persons is similar to that used for other febrile neutropenic patients, such as solid tumor patients receiving myelotoxic chemotherapy. First, an empirical regimen of antibiotics should be selected, based on the pattern of bacterial susceptibility and nosocomial infections in the particular institution and the degree of neutropenia. Broad spectrum empiric therapy with high doses of one or more antibiotics should be initiated at the onset of fever. Aminoglycosides should be avoided whenever feasible due to associated toxicities. Therapy should be continued until the patient is afebrile for 24 hours and the absolute neutrophil count (ANC) is greater than or equal to 0.5×10^9 cells/l (500 cells/uL). Combination regimens often prove to be more effective than monotherapy. The potential for additivity or synergy should be present in the choice of antibiotics.

(2) Modifications of this initial antibiotic regimen should include a thorough evaluation of the history, physical findings, laboratory data (including chest radiograph), and epidemiological information. Antifungal coverage with amphotericin B should be added, if indicated, for patients who remain persistently febrile for 7 days or more on antibiotic therapy in association with clinical evidence of infection, or if they have new fever on or after day seven of treatment with antibiotics. If there is evidence of resistant gram-positive infection, vancomycin should be added. If diarrhea is present, stool cultures should be examined for Salmonella, Shigella, Campylobacter, and Yersinia. Oral and pharyngeal mucositis and esophagitis suggest herpes simplex infection or candidiasis. Empiric antiviral and/or antifungal therapy should be considered.

(3) Surveillance cultures may be useful for monitoring acquisition of resistant bacteria during prophylaxis and emergence of fungi. A once or twice weekly sampling of surveillance cultures from natural orifices and skin folds (for example, axillae, groin) would be reasonable, but should be modified based on the institutional patterns of nosocomial infections. A chest radiograph should be considered at initiation of empiric therapy. This may aid in definitive diagnosis of a new pulmonary infiltrate obtained during the course of neutropenia. The principles described above are generally applicable to the febrile neutropenic patient and provide a foundation upon which a specific initial regimen may be selected. These principles are summarized as follows:

- Principle 1: The spectrum of infecting organisms and antimicrobial susceptibility patterns vary both among institutions and over time.

- Principle 2: Life-threatening, gram-negative bacterial infections are universal among neutropenic patients, but the prevalence of life-threatening, gram-positive bacterial infections varies greatly among institutions.

- Principle 3: Current empiric antimicrobial regimens are highly effective for initial management of febrile, neutropenic episodes.

- Principle 4: Search for the nidus of infection, that is, look for the reason the patient is infected, and eliminate it.

Overall recommendations for managing infections are summarized as follows:

- A standardized plan for the management of febrile, neutropenic patients must be devised.

- Empiric regimens must contain antibiotics broadly active against gram-negative bacteria, but antibiotics directed against gram-positive bacteria need be included only in institutions where these infections are prevalent.

- No single antimicrobial regimen can be recommended above all others, as pathogens and susceptibility vary with time.

- If infection is documented by cultures, the empiric regimen may require adjustment to provide appropriate coverage for the isolate. This should not narrow the antibiotic spectrum.

- When the patient remains afebrile, the initial regimen should be continued for a minimum of 7 days.

d. Immune Globulin Administration. Immune globulins have not been shown to be beneficial for radiation casualties on a general basis. However, their use may be beneficial in specific diseases. Passive intravenous administration of immunoglobulins (Ig) following acute radiation injury is a therapeutic measure employed to counter the suppressive effects of radiation on the immune system and to diminish susceptibility to infection or its pathologic consequences. The therapeutic strategies for employing the Ig treatments fall into two, quite distinct camps. The first, more commonly applied strategy (in total body irradiated patients receiving bone marrow transplants) involves periodic blood infusions in an attempt to bolster the diminished Ig blood plasma levels that are critical in combating challenge by a variety of infectious agents. The second strategy, which is more experimental in nature and less certain in terms of efficacy, is designed to selectively control the pathogenic responses related to septic shock and associated overexpression of inflammatory cytokines.

e. Other Considerations.

(1) *Source of infection.* As with any infection, primary consideration should be locating the nidus of infection and if possible, eliminating it. Central venous and indwelling catheters must be considered liabilities in these immunocompromised patients. Other sites of possible infections should not be forgotten and teeth, sinuses, and urine commend special attention.

(2) *New adjuvant therapies.* Sepsis and its sequelae are the major causes of death in severely irradiated patients. This is also true of patients in medical and intensive care units in the US. In spite of an intense research effort toward identifying causative inflammatory mediators and therapeutic protocols for the treatment of this lethal condition, relatively few advances have occurred. In recent years, several candidate therapies, including immunomodulators, corticosteroids, antiendotoxin antibodies, and inflammatory cytokine antagonists, have been evaluated. While significant insights have been realized into the pathophysiology of sepsis, none of the clinical trials to date have achieved statistically significant reductions in mortality according to the prescribed criteria for efficacy.

(3) *Radiation factors.* Consideration must be given to the type of radiation exposure (internal or external), the presence of non-hematopoietic organ injury, and the radioisotope. Growth factor therapy may not be helpful and may be detrimental where there is prolonged internal irradiation by long lived isotopes. Potential risks include acceleration of leukemogenesis and prevention of neutrophil migration into soft tissues. Early granulocyte recovery following growth factor therapy may indicate a lower than estimated radiation dose.

(4) *Antibiotic-induced release of endotoxins.* Endotoxins, the lipopolysaccharide components of gram-negative bacterial cell membranes, are potent inducers of the proinflammatory cytokines, including tumor necrosis factor (TNF) and IL-1. They are known to play a pivotal role in the pathogenesis of sepsis. Experimental models and clinical studies have shown that endotoxin concentration in the blood and urine can increase following antibiotic treatment of gram-negative infections. Antibiotics differ in their capacity to cause endotoxin release from gram-negative bacteria. Preclinical studies suggest that this effect may translate into increased morbidity and mortality. The clinical relevance of these differences between antibiotics in endotoxin-releasing potential is unknown.

(5) *Granulocyte transfusions from cytokine-stimulated donors.* Extensive literature is available regarding the therapeutic and prophylactic efficacy of transfusing granulocytes (polymorphonuclear neutrophils [PMNs]) into neutropenic individuals. Cell dose and alloimmunization have limited the applicability and evaluation of granulocyte transfusions (GTX). The use of G-CSF to significantly increase the number of donor circulating PMNs over a time duration that would allow multiple sampling may help alleviate this problem. The use of human leukocyte antigen (HLA)-compatible donors may avoid the problem of alloimmunization. Filgrastim provides an additional benefit, as it enhances the phagocytic and microbicidal activity of stimulated PMNs. Granulocyte-colony stimulating factor, when administered to normal individuals, significantly improves the quantity of granulocytes collected, which results in significant circulating levels of granulocytes in neutropenic patients. Granulocyte transfusions of G-CSF-stimulated PMNs could prove effective therapy for severely neutropenic patients with sepsis and who have failed to respond to appropriate antibiotic therapy. Patients with prolonged, severe neutropenia caused by continuing bone marrow failure, as well as those with refractory infection, may benefit.

f. Selective Decontamination of the Digestive Tract. Selective decontamination of the digestive tract has not led to fewer febrile episodes or to a lower mortality in neutropenic patients. Therefore, this procedure should be considered experimental and cannot be

recommended as a routine preventive strategy. However, the patient who may benefit from an intensive, oral nonabsorbable antibiotic regimen is one who is likely to experience prolonged, profound granulocytopenia and who may have some degree of mucosal damage. Quinolones can provide elimination of potentially pathogenic aerobic gram-negative bacilli from the GI tract and prevent colonization and subsequent infection. The noted efficacy of quinolones in decreasing the frequency of gram-negative infections is clouded by the inability to attribute the efficacy to selective gut decontamination or increased tissue levels of the drugs. Trimethoprim-sulfamethoxazole has been known to cause idiopathic marrow suppression and may be detrimental in the face of irradiation injury.

g. Hematopoietic Growth Factors. Hematopoietic growth factors, such as G-CSF (filgrastim) and GM-CSF (sargramostim), are potent stimulators of hematopoiesis (see Appendix C). Preclinical studies in nonhuman subjects have demonstrated a significant reduction in the duration of neutropenia. The time to recovery of neutrophils to preirradiated values is markedly reduced. The risk of infection and subsequent complications are directly related to depth and duration of neutropenia. In severe radiation-induced myelosuppression, where clinical support in the form of antibiotics and fresh, irradiated platelets or whole blood is used concurrently with G-CSF or GM-CSF, a marked reduction in infectious complications translates to reduced morbidity and mortality. The beneficial role of G-CSF or GM-CSF in reducing the duration and degree of neutropenia cannot be underestimated. It has been determined that the longer the duration of severe neutropenia, the greater the risk of secondary infections, especially with invasive mycoses. An additional benefit of the cytokines is their ability to increase the functional capacity of the neutrophil and thereby contribute to the prevention of infection as an active part of cellular host defense.

(1) *Initiation and duration.* In order to achieve maximum clinical response, G-CSF or GM-CSF should be started within 24 to 72 hours subsequent to the exposure. This provides the opportunity for maximum recovery. Cytokine administration should continue, with daily consecutive injections, to reach the desired effect of an ANC of 1.0×10^9/l after to the ANC nadir. Cytokine recommendations for patients who are expected to experience severe levels of febrile neutropenia are as follows:

- Recommended administration is once daily subcutaneously or intravenously (IV).

- Granulocyte-colony stimulating factor (filgrastim): 2.5 to 5.0 µg/kg/day (100-200 µg/m ^{2}/day); subcutaneously.

- Granulocyte macrophage-colony stimulating factor (sargramostim): 5.0 to 10.0 µg/kg/day (200-400 µg/m ^{2}/day); subcutaneously.

(2) *Therapeutic efficacy*. Although reports are available that suggest the delay of cytokine administration did not compromise recovery of neutrophils, other data suggest that delay of cytokine administration results in loss of therapeutic efficacy. The lack of a clear beneficial effect relative to cytokine scheduling may be due, in part, to the variable degrees of

myelosuppression used in the preclinical and clinical studies available. Schedule variation may be less well tolerated in the cases of severe myelosuppression.

(3) *Comparative toxicity of cytokines.* The predominant side effect noted with administration of G-CSF (filgrastim) is medullary bone pain, which may be observed shortly after initiation of G-CSF treatment, and again just before onset of neutrophil recovery from nadir. The G-CSF may exacerbate preexisting inflammatory conditions. The most noted side effects with administration of GM-CSF (sargramostim) are fever, nausea, fatigue, headache, bone pain, and myalgia. It is not clear whether side effects of G-CSF or GM-CSF differ markedly when conventional doses are administered.

h. Thrombocytopenia and Anemia.

(1) *Conventional therapy of thrombocytopenia.* The requirement for platelet support depends on the patient's condition. In irradiated patients with or without other major medical problems (infection, GI problems, or trauma), the platelets should be maintained at greater than 20×10^9/L. Analysis of platelet counts versus hemorrhage suggests that 10×10^9/L is adequate in the absence of any indication of accompanying frank hemorrhage. If surgery is needed, the platelet count should be greater than 75×10^9/L. Transfusion of platelets remains the primary therapy to maintain adequate platelet counts. As general supportive measures, one should avoid the use of aspirin and nonsteroidal, anti-inflammatory drugs. Limited platelet support is likely to come from random donors. Should refractoriness develop, family members as well as HLA-compatible donors from the general population can be considered as platelet donors. The use of platelet products from which white blood cells have been removed is desirable to minimize both allosensitization and the risk of transmission of viral illnesses, such as cytomegalovirus. All blood products should receive 15 to 20 Gy of radiation before infusion to prevent graft-versus-host disease through infusion of mononuclear cells present in the products. If an allotransplant is contemplated, the use of platelets from related donors should be avoided.

(2) *Growth factor/cytokine therapy for thrombocytopenia.* Use of thrombopoietic agents immediately after radiation injury is probably appropriate under the conditions of symptomatic thrombocytopenia or hemorrhage. Further drug development may alter the accepted pattern of care. The synthetic IL-3 receptor agonist Synthokine and megakaryocyte growth and development factor/thrombopoietin (MGDF/Tpo) may be appropriate in the post-irradiation phase, particularly if combined injury has occurred.

(3) *Conventional therapy of anemia.* Transfusion of peripheral red blood cells (PRBCs) remains the primary therapy to maintain hemoglobin above 8 gm/dl. Peripheral red blood cells transfusions should be irradiated, leukocyte-filtered (whenever possible), and from an unrelated donor if allogeneic transplantation is a consideration. Risks of PRBC transfusion may include cytomegalovirus (CMV) transmission and alloimmunization. Gamma irradiation of blood products with 20 Gy will diminish graft versus host reactions common in radiation casualties.

(4) *Erythropoietin therapy of anemia.* Use of erythropoietin (Epo) after radiation injury is not recommended even though it is likely to be safe. Anemia is not generally

life-threatening in this situation; endogenous Epo levels are often already elevated after highly cytotoxic therapy and evidence of benefit is not yet available from clinical chemotherapy models.

i. Bone Marrow Transplant. The dilemma presented to the physician is whether or not a transplant is required to ensure the best chance of survival for the severely irradiated person. Increasing doses lead to more protracted pancytopenia and increasing risk of death from infection and/or hemorrhage. The dose of radiation to the bone marrow that would effectively prevent recovery within a period required for survival is unknown; however, this dose can be effectively increased with the use of conventional clinical support and cytokine therapy. Preclinical studies using a canine model of lethal whole body irradiation have shown that therapeutic support (including fluids, platelets or whole blood, antibiotic regimens, and administration of G-CSF) has induced survival in otherwise uniformly LDs of radiation.

(1) Bone marrow transplant may be beneficial in those patients whose dose estimate approaches the $LD_{50/60}$ for humans receiving clinical support, or 6 Gy free-in-air for whole body uniform exposure. Human exposures due to accident or in a tactical environment are unlikely to be whole body uniform exposure; that is, only part of the marrow will be exposed. The remainder will receive a lower dose, or not be exposed at all. The use of broad spectrum antibiotics, blood product support, and hematopoietic growth factors may provide a human a reasonable chance of surviving a radiation dose of 8 Gy or greater.

(2) The major complications following autologous bone marrow transplantation (ABMT) are the same as those consequent to severe radiation exposure. Obligate periods of neutropenia and thrombocytopenia present the increased risk for infection and hemorrhage, the need for platelet and/or red blood cell (RBC) transfusions and delayed or incomplete reconstitution, and potential "other organ" damage. Allogeneic bone marrow transplant (allo-BMT) also presents the risk of graft-versus-host disease and graft rejection. There has been no evident increase in complications of graft-versus-host disease, graft rejection, or relapse associated with cytokine use in randomized studies of allo-BMT. Patients being considered for allo-BMT must meet the following conditions:

- A fully matched sibling donor is available.
- The patient has an absolute lymphocyte count < 1.0 10^9 /l.
- The radiation dose is unknown but is likely to be between 6 and 20 Gy.
- Irradiation is not ongoing from an internal source.
- There are no other injuries or diseases that preclude survival or preclude transplantation (severe burns).

(3) The timing of marrow grafting is crucial and presents a dilemma. Experimental data suggest that the marrow should be infused within the first 3 to 5 days of radiation exposure. This coincides with the peak period of immunosuppression; and graft rejection, therefore, will be less likely. These findings stress the importance of developing reliable clinical and laboratory parameters to assess the degree of radiation damage to the marrow as quickly as possible and to determine which patients should be given marrow transplants. Waiting for a week or longer after the radiation exposure would require some form of immunosuppressive treatment to prepare the patient for a marrow graft because such treatment may be less tolerated by the patient who is a radiation accident victim. Timing of the marrow graft should also be viewed in the context of recommended cytokine therapy for the neutropenic patient. Assuming all indications point toward a high radiation exposure, it is recommended that cytokine therapy be initiated as soon as possible. In this case, the patient would have been treated with G-CSF or GM-CSF prior to the decision to transplant. In addition, further drug development and results from ongoing clinical trials may alter the recommendations with the introduction of thrombopoietic agents such as IL-6, IL-11, PIXY-321, MGDF/Tpo, or Synthokine. The effects of prior cytokine administration on marrow engraftment are unknown.

(4) The same doses, routes, and schedules of cytokine administration (G-CSF or GM-CSF) as mentioned previously for the neutropenic patient should be followed in the transplant setting.

(5) The occurrence of delayed or inadequate primary marrow engraftment or secondary graft failure following autologous or allogeneic stem cell transplantation can be treated with cytokine administration and/or peripheral blood progenitor cell transfusion. An attempt at cytokine therapy is reasonable in view of the high infectious mortality rates in this population and the general desperation inherent in this situation. Only limited data is available assessing the value of cytokines in stimulating recovery and improving survival of patients with poor engraftment after transplant.

(6) There is increasing evidence that peripheral blood stem cell transplanation (PBSCT) of cells mobilized by growth factors are capable of reliable, rapid, and durable autologous hematopoietic engraftment. An early assumption is that autologous mobilized (primed) PBSCT offered more rapid recovery of granulocytes and platelets than BMTs derived from normal, resting marrow.

(7) The critical number of allo-CD34+ cells from growth factor mobilized blood to ensure a durable graft is unknown. An optimal autologous PBSCT CD34+ cell number is in the range of 2-5 x 10^6 per kg body weight. The estimate for allo-PBSCT CD34+ cells is in the range of 5-15 x 10^6 per kg. This should be achievable with two apheresis.

(8) Cryopreserved, mobilized peripheral blood stem cells (PBSCs) have been successful in the treatment of graft failure. It is reasonable that allo-PBSCs be harvested and cryopreserved in the event of allo-BMT failure to engraft or secondary graft failure. Efforts should also proceed to evaluate the engraftment potential of primed bone marrow derived from the GF-stimulated donor at the approximate time of PBSC harvest by apheresis.

(9) Further consideration must also be given to the combination transplant protocol of PBSC plus BM transplantation. Such a protocol would potentially take advantage of the dual engraftment properties available in PBSC grafts. The mobilized peripheral blood cells contain large quantities of committed progenitors in addition to hematopoietic stem cells. These committed progenitors would provide for an earlier, although unsustained, phase of engraftment. The more primitive stem cells contained in both the PB and BM graft would then provide for the later, durable, long-term reconstitution.

(10) The predominant use of G-CSF (filgrastim) for mobilization of PBSCs is in the dose range of 2 to 16 μg/kg/day. The higher doses have been administered as a divided dose, subcutaneously, twice a day for 5 or 6 consecutive days. The lower doses have been administered once a day, subcutaneously, for the same duration. Leukapheresis can be performed for 2 consecutive days beginning on day 5 of G-CSF administration. The specific protocol and dose of G-CSF should be the choice of the transplant team. The choice of cytokine as a single agent or in combination with other GFs should also be the choice of the transplant team, based on experience and current knowledge of GF-induced mobilization efficiency. The cytokines GM-CSF, c-kit ligand, erythropoietin, and IL-3 have been used both alone or in combination to induce PBSC mobilization. It is probable that the flt-3 ligand, and the IL-3 receptor agonist daniplestim, will be evaluated for mobilization potential.

6-22. Therapeutic Support for the Severely Irradiated Patient: Gastrointestinal Syndrome.

a. The effects of radiation on the GI tract and the associated symptomatology can be categorized into four major phases that correspond to the elapsed time from exposure to manifestation. These phases are--

- **The Prodromal Phase**, in which nausea, vomiting, and diarrhea occur minutes to hours after exposure.
- **The Subacute Phase**, in which diarrhea and vomiting occur hours to days after exposure.
- **The Acute Phase**, in which diarrhea, toxemia, and septicemia occur days to weeks after exposure.
- **The Chronic Phase**, in which survivors may develop fibrosis, bleeding, and fistulas months to years after exposure.

b. Nausea and vomiting associated with the prodromal effects of radiation exposure can be prevented and/or ameliorated by the new generation of 5-HT_3-receptor antagonists such as ondansetron and granisetron. During the subacute and acute phases, fluids and electrolytes should be administered to prevent or correct dehydration. If blood transfusions are administered, the blood should be irradiated to diminish graft versus host reactions.

c. A number of factors may contribute to radiation-induced diarrhea. Diarrhea associated with the prodromal and subacute phases of GI injury is most likely related to neurohumoral factors affecting GI motility and transport. Loss of the epithelial cell lining is not observed until later during the acute phase of GI injury. As a result, treatment for postirradiation diarrhea will require several different approaches. For the early prodromal and subacute phases of diarrhea, agents directed against, or counteracting the effects of neurohumoral factors on GI cells should be considered. These include antidiarrheal/antisecretory agents such as anticholinergics, metamucil, amphogel, and loperamide. Loperamide may offer distinct advantages as the drug affects both intestinal cell transport and motility, each of which may contribute to diarrhea. Antisecretory agents, however, will be of limited effectiveness against the acute phase of GI injury, during which the loss of epithelial cell lining has progressed to denudation of the intestine.

d. Presently, the level of understanding concerning the processes involved in stimulating proliferation and/or maintaining the intestinal cell lining following radiation exposure prevents the recommendation of specific therapies for the acute phase of GI injury. Cytokines that have been proven to be effective in promoting recovery of hematopoietic injury may be beneficial to the intestine as well. Sufficient data concerning the efficacy of these agents on gut-related growth factors and elemental diets in stimulating GI regeneration are not yet available.

e. The use of antibiotics should be considered for specific infections. Gut decontamination has been recommended following hematopoietic injury, however, the potential adverse effects of gut decontamination also require consideration. Because of the complex relationship between the gut and the immune system, gut decontamination may eliminate the potentially beneficial effects derived from the normal flora in stimulating immune defenses and preparing the immune system for subsequent infections. In the future, the capability to maintain intestinal integrity following radiation exposure may reduce the present emphasis on gut decontamination.

f. The bactericidal effect of gastric acid on intestinal flora is well known. However, gastric acid also stimulates pancreatic and biliary secretions, both of which have adverse effects on postirradiation GI integrity. Reduction of gastric acidity may be beneficial in the GI syndrome. Thus, the need to maintain gut integrity may preempt the desire to stimulate normal bactericidal mechanisms by increasing gastric acid secretion. Suppression of gastric acid secretion can be accomplished by the administration of Histamine-2 antagonists (for example, cimetidine) or H^+,K^+ ATPase inhibitors (omeprazole). However, the efficacy of these agents in irradiated patients is unproven and they may also diminish normal bacterial flora.

6-23. Summary of Medical Aspects of Acute Radiation Injury

Tables 6-10 through 6-13 summarize the current ideas on the treatment of radiation casualties at progressively increasing dose levels. The treatment modalities are meant as guidelines for medical officers during war conditions where nuclear weapons have been utilized. As such, they are not substitutions for service or joint regulations applicable to specific contingencies and also

should not be construed as to manipulate national occupational radiation exposure limitations or limits established for low level radiation incidents (see Section X). It should be noted that RES categories and corresponding dose estimates in these tables are for comparison purposes only. Also, RES is a collective/unit dose designator and is not assigned against an individual. See STANAG 2083.

Table 6-10. Medical Aspects of Radiation Injury (0 to 3 Gy)

DOSE (estimate) Unit Radiation Exposure Status (RES)	INITIAL SYMPTOMS	INITIAL SYMPTOMS INTERVAL ONSET - END	ANTIEMETIC PRETREAT-MENT EFFECT	MEDICAL PROBLEMS	INDICATED MEDICAL TREATMENT	DISPOSITION WITHOUT MEDICAL CARE	DISPOSITION WITH MEDICAL CARE	CLINICAL REMARKS
0 - 0.35 Gy --- RES 1	None	N/A	Dry mouth Headache	Anxiety	Reassurance. Counsel at redeployment	duty	duty	Potential for combat anxiety manifestation
0.35 - 0.70 Gy --- RES 1	Nausea, mild headache	ONSET 6 hrs END 12 hrs	Not determined	Anxiety	Reassurance. Counsel at redeployment	duty	duty	Mild lymphocyte depression within 24 hours
0.70 - 1.25 Gy --- No further radiation exposure allowable RES 2	Transient mild nausea, vomiting in 5 - 30% of personnel	ONSET 3 - 5 hrs END 24 hours	5 - 30% of personnel nauseated without emesis	Potential for delayed traumatic and surgical wound healing, minimal clinical effect	Debridement and primary closure of any and all wounds. No delayed surgery.	Restricted duty No further radiation exposure elective surgery or wounding	restricted duty No further radiation exposure	Moderate drop in lymphocyte, platelet, and granulocyte counts. Increased susceptibility to opportunistic pathogens.
1.25 - 3.0 Gy --- RES 3	Transient mild to moderate nausea and vomiting in 20 - 70% of personnel. Mild to moderate fatigability and weakness in 25 - 60% of personnel	ONSET 2 - 3 hrs END 2 days	Decreased vomiting, Possible increase of fatigability	Significant medical care may be required at 3 - 5 wks for 10 - 50% of personnel. Anticipated problems should include infection, bleeding, and fever. Wounding or burns will geometrically increase morbidity and mortality	Fluid and electrolytes for GI losses Consider cytokines for immunocom-promised patients (follow granulocyte counts)	LD_{5} to LD_{10} Restricted duty No further radiation exposure, elective surgery or wounding. May require delayed evacuation from theater during nuclear war IAW command guidance.	Restricted duty No further radiation exposure, elective surgery or wounding	If there are more than 1.7×10^{9} lymphocytes per liter 48 hrs after exposure, it is unlikely that an individual has received a fatal dose. Patients with low (300-500) or decreasing lymphocyte counts, or low granulocyte counts should be considered for cytokine therapy and biologic dosimetry using metaphase analysis where available

Table 6-11, Medical Aspects of Radiation Injury in Nuclear War (3 to 5 Gy)

DOSE (estimate) ---------- - Unit Radiation Exposure Status (RES)	INITIAL SYMPTOMS	INITIAL SYMPTOMS INTERVAL ONSET - END	ANTIEMETIC PRETREAT-MENT EFFECT	MEDICAL PROBLEMS	INDICATED MEDICAL TREATMENT	DISPOSITION WITHOUT MEDICAL CARE	DISPOSITION WITH MEDICAL CARE	CLINICAL REMARKS
3.0 - 5.0 Gy ---------- RES 3	Transient moderate nausea and vomiting in 50 - 90% of personnel. Early: Mild to moderate fatigability and weakness in 80 - 100% of personnel	Nausea / vomiting ONSET 2 hrs END 3 - 4 days Diarrhea ONSET at 10 days END 2 - 3 wks	Undetermined	Frequent diarrheal stools, anorexia, increased fluid loss, ulceration, death of crypt cells and Peyer's Patch lymphoid tissue Increased infection susceptibility during immuno-compromised time frame Bleeding diathesis at 3 - 4 wks due to megakaryocyte loss	Fluid and electrolytes for GI losses Consider cytokines for immunocom-promised patients (follow granulocyte counts) Specific antibiotic therapy for infections May require GI decontamination with quinolones, use alimentary nutrition	LD_{10} to LD_{50} Survivors may be able to return to light duty after 5 weeks. No further radiation exposure. May require delayed evacuation from theater	Increased percentage of survivors may be able to return to duty after 5 weeks. No further radiation exposure. May require evacuation from theater for adequate therapy.	Moderate to severe loss of lymphocytes. Follow counts q6h in first few days if possible for prognosis. Moderate loss of granulocytes and platelets. Hair loss after 14 days, Thrombocytopenic purpura appears after 3 weeks. Consider cytokine therapy and biologic dosimetry using metaphase analysis where available Loss of crypt cells and GI barriers may allow pathogenic and opportunistic bacterial infection. Use alimentary nutrition to encourage crypt cell growth. Avoid parenteral nutrition and central intravenous lines. Anticipate anaerobic colonization. All surgical procedures must be accomplished in initial 36-48 hrs after irradiation. Any additional surgery must be delayed until 6 wks post exposure.

Table 6-12. Medical Aspects of Radiation Injury in Nuclear War (5 to 8 Gy)

DOSE (estimate) ---------- -- Unit Radiation Exposure Status (RES)	INITIAL SYMPTOMS	INITIAL SYMPTOMS INTERVAL ONSET - END	ANTIEMETIC PRETREAT-MENT EFFECT	MEDICAL PROBLEMS	INDICATED MEDICAL TREATMENT	DISPOSITION WITHOUT MEDICAL CARE	DISPOSITION WITH MEDICAL CARE	CLINICAL REMARKS
5.0 - 8.0 Gy ---------- -- RES 3	Moderate to severe nausea and vomiting in 50 - 90% of personnel. Early: Moderate fatigability and weakness in 80 - 100% of personnel, frequent diarrhea	ONSET under 1 hr END indeterminate, may proceed directly to GI syndrome without a break	None	At 10 days to 5 weeks, 50 to 100% of personnel will develop pathogenic and opportunistic infections, bleeding, fever, loss of appetite, GI ulcerations, bloody diarrhea, nausea, severe fluid and electrolyte shifts, third space losses, capillary leak, hypotension	Tertiary-level intensive care required to improve survival. Fluid and electrolytes for GI losses, may require transfusion and/or colloids Cytokines for immunocom-promised patients Specific antibiotic therapy for infections, to include antifungals Will require GI decontaminatio n with quinolones, use alimentary nutrition	LD_{50} to LD_{90} At low end of exposure range, death may occur at 6 wks in more than 50% At high end of exposure range, death may occur in 3 - 5 wks in 90%	Early evacuation to tertiary-level medical center before onset of manifest illness. Patients will require extensive reverse isolation to prevent cross contamination and nosocomial infection.	Practically no lymphocytes after 48 hrs. Severe drop in granulocytes and platelets later. In pure radiation exposure scenarios, these patients will require highest priority evacuation. The latent period between prodromal symptoms and manifest illness may be very short. When this radiation injury is combined with any significant physical trauma, survival rates will approach zero. All surgical procedures must be accomplished in initial 36-48 hrs after irradiation. Any additional surgery must be delayed until 6 wks postexposure. Partial marrow shielding may complicate bone marrow transplant. Steroid therapy is ineffective

Table 6-13. Medical Aspects of Radiation Injury in Nuclear War (8-30+ Gy)

DOSE (estimate) ---------- --- Unit Radiation Exposure Status (RES)	INITIAL SYMPTOMS	INITIAL SYMPTOMS INTERVAL ONSET - END	ANTIEMETIC PRETREAT-MENT EFFECT	MEDICAL PROBLEMS	INDICATED MEDICAL TREATMENT	DISPOSITION WITHOUT MEDICAL CARE	DISPOSITION WITH MEDICAL CARE	CLINICAL REMARKS
8.0 - 30+ Gy ---------- --- RES 3	Severe nausea, vomiting, fatigability, weakness, dizziness, and disorientation. Moderate to severe fluid and electrolyte imbalance, hypotension, possible high fever, and sudden vascular collapse.	ONSET less than 3 minutes END death	None	LD_{100} at 10 Gy death at 2-3 wks. Minimal if any break between prodromal syndrome and manifest illness. At high radiation levels, CNS symptoms predominate, with death secondary to cerebral vascular incompetence	Supportive therapy in higher dosage ranges. Aggressive therapy if pure radiation injury and some evidence of response	LD_{90} to LD_{100} Expectant category	If assets are available, then early evacuation to tertiary-level medical center during manifest illness. Patients will require extensive reverse isolation to prevent cross contamination and nosocomial infection. Most patients will remain expectant.	Bone marrow totally depleted within days. Bone marrow transplant may or may not improve ultimate outcome, due to late radiation pneumonitis and fibrotic complications. Even minor wounds may prove ultimately fatal. Aggressive therapy is indicated when resources are available and transport to a tertiary care medical center is possible.

Section V. COMBINED INJURY--RADIATION, TRAUMA, CHEMICAL, AND BIOLOGICAL WEAPONS

6-24. General

A nuclear explosion results in a multiplicity of traumas like thermal, chemical, and biological as well as injuries from the blast or high velocity metal or glass or from deceleration or blunt trauma. Only about a third of these casualties are likely to have solitary injures. Two-thirds of the casualties will have combinations of injuries from the detonation. Because the blast, thermal, and radiation envelopes occur over roughly the same area, combined injury will be the norm. Only a small percentage of physical trauma casualties will not have some form of radiation injury that will complicate their recovery (see Table 6-14).

Table 6-14, Predicted Distribution of Injuries Sustained from a Nuclear Explosion

Injury Types	Percentage of Total Injuries
Combined Injuries	**65 to 70**
Irradiation, Burns, and Wounds	20
Irradiation and Burns	40
Irradiation and Wounds	5
Wounds and Burns	5

6-25. Hematopoietic Effects of Combined Injury

a. Immunological Effects. Radiological injury significantly compounds the morbidity and mortality of other injuries by compromising the integrity of the immune system. Therefore, early healing and damage control systems rapidly deplete reserves that are then unable to regenerate due to the radiation injury. Little research has been performed on the combined effects of weapons of mass destruction. As radiological injury predisposes a casualty to infection, a logical assumption can be inferred that biological weapons will be devastating against a previously radiologically injured populace. A higher rate of infection and a more virulent disease course would be anticipated in this scenario. Combined injury patients in this category will require early evacuation from theater.

b. Pancytopenia. Since reserves are depleted and consumed without adequate regeneration, pancytopenia develops more rapidly than in the pure radiologically injured patient. Acute blood loss that occurs as a result of a physical trauma cannot be replenished by increased marrow output. Likewise, megakaryocytes are unable to replicate and platelets are consumed. Lack of release of new erythrocytes complicates anemia by the aging of the red cell population and subsequent decrease in oxygen carrying ability. Fibroblasts that are damaged by irradiation do not replicate at a normal rate, and effects of infection are magnified by the immuno-suppression (see Table 6-15).

Table 6-15. Hematopoietic Effects of Combined Injury.

Radiation	Trauma
Anemia	Depletion of vascular reserves.
Bleeding	Abnormal clotting; increased viscosity.
Infection	Consumption of marrow progenitors.

6-26. Burns and Radiation

a. Thermal Burns. Experimental data demonstrate that the mortality of thermal burns markedly increases with irradiation (see Figure 6-2). Burns with 50 percent mortality may be transformed into more than 90 percent mortality by concomitant radiation doses as small as 1.5 Gy. The findings in Figure 6-2 are from animal studies using a rat model that is almost twice as radioresistant as man. At 2.5 Gy (250 rads), no animals die. If they concomitantly receive a significant burn, then nearly all die. This level of radiation is approximately equal to the standard military guidance that 1.5 Gy is the maximum acceptable risk dosage for any military operation. At this level, a unit is considered fully combat effective but is not to be exposed to further radiation. A unit exposed to this level is described as RES 3 (see Table 6-3).

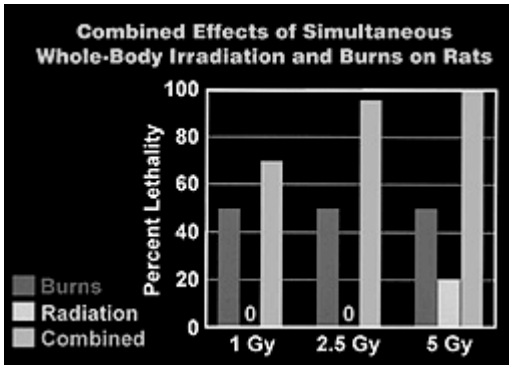

Figure 6-2. Experimental data on combined radiation and burn injury.

b. Treatment. The primary treatment of significant radiation injury includes reduction of the nidus of infections. Full thickness burns are an ideal bacterial culture media, and excision of these burns may be indicated to allow primary closure. No changes should be made in the indications for escharotomy. No studies are available regarding the use of modern skin graft techniques in irradiation injury victims. Use of topical antimicrobials (such as silver sulfadiazene) whose side effects include leukopenia may be a complicating factor in radiation immunocompromised patients. No data are available regarding the response to clostridial infection, and strong consideration should be made as to the use of passive tetanus immunization even in previously immunized patients. Early excision and grafting of the full thickness burn allows wound closure at the earliest point in time. No data truly exists for the aggressive

management of partial thickness burns in the irradiated patient. Stimulation of the marrow with cytokines has not been used in burn patients but may prove efficacious and may also stimulate skin regeneration. The firemen at Chernobyl did not do well despite aggressive therapy including bone marrow transplant. Patients whose burns are contaminated by radioactive material should be gently decontaminated to minimize absorption through the burned skin. Most radiological contaminants will remain in the burn eschar when it sloughs. Key treatment protocols are summarizes as follows:

- Elimination of infection reservoirs by debridement of , covering, and using microencapsulated antibiotics.

- Support of immune mechanisms.

- Use of antimicrobials such as Mafenide Acetate cream and Silver Sulfadiazine.

- Suppression of infection transfer.

6-27 Wounds and Radiation

Most patients will arrive at MTFs in varying states of urgency. Most survivors of combat trauma have extremity wounds. Those with devastating thorax and head wounds often do not survive to become patients. If air evacuation is not feasible, then fewer still will arrive at the supporting hospital. The presence of forward surgical teams (FSTs) may make a positive impact on the case mix. In the case of a nuclear detonation, much of the team's work will be quick-patch-and-hold in order to diminish overall morbidity by sacrificing more tenuous patients.

a. Patients who have compromised immune mechanisms are more likely to have even clean wounds become infected. Military wounds are not clean to begin with, and, in the face of wound infection, dehiscence is common; and if fibroblasts cannot adequately bridge the wound, closure fails. If the patient survives, the wound will be very slow to heal. Wounds from intense local irradiation heal extremely slowly, and graft failures over radiogenic ulcerations usually fail. Slow extremity wound healing may require amputation in order to prevent systemic deterioration. The decision to amputate an extremity that in ordinary circumstances would be salvageable will rest with the surgeon in the first two days following the combined injury. Little data is available in regard to healing mechanisms in the wound contaminated with radionuclides. Obviously they should be removed as with any other foreign material. Once closed in the wound, absorption and systemic distribution will occur.

b. If at all possible, wounds should be closed primarily as early as possible. Extensive debridement of wounds may be necessary in order to allow this closure. Traditionally, combat wounds are not closed primarily due to the high level of contamination, devitalized tissue, and the subsequent morbidity and mortality of the closed space contamination. In the case of the radiation combined injury patient, aggressive therapy will be required to allow survival. Early wound repair eliminates the nidus of infection and may cause early stimulation

of the marrow by the wound healing mechanisms. No studies are available regarding the use of aggressive marrow resuscitation as outlined in previous sections in the physically wounded patient. Also, a second procedure will induce additional trauma and consumption of hematological reserves.

c. A brief summary of combined wound and radiation treatment protocols are as follows:

- Stopping hemorrhage must come first.

- Debridement of all questionable tissue and foreign material must occur. Tissue that is not definitely viable should be removed.

- Any structures vital to maintenance of the extremity or viability must be repaired as early as possible.

- Irrigation with saline can be used to cleanse wounds. Nonionic cleansing liquids should also be safe.

d. The following subsections describe considerations for specific critical tissues and organs:

(1) *Vascular*. There is no experience using modern surgical techniques in the management of vascular injury in the combined injury patient. The best recommendations are to avoid protocols that include the necessity for secondary surgeries, and to adhere to the following:

- Ligation.

- Primary repair.

- Autologous graft.

- Synthetic graft.

(2) *Bowel*. Bowel surgical indications appear to remain unchanged. Takedown of temporary exteriorizations should be delayed until complete recovery from the radiological injury has occurred.

- Small bowel: Repair and exteriorize.

- Large bowel: Be selective in management of the wound and exteriorize all injuries.

- Colostomy revisions: Only after complete hematopoietic recovery (three months or more).

(3) *Spleen*. As the spleen is a significant leukocyte reservoir, salvage may be important in the long-term recovery of the patient. There is no evidence that splenic neo-hematopoiesis occurs in humans. Other considerations are to--

- Make the maximum effort to repair.
- Conduct a partial splenectomy (splenic bed must be dry).
- Surgical indications remain the same as in standard treatment protocols..

e. Early work in radiation and surgery has not been validated for use with aggressive therapeutic management of radiation injury. The data available demonstrate that timing of wounding and/or surgery directly impacts surgical morbidity and mortality (see Figure 6-3).

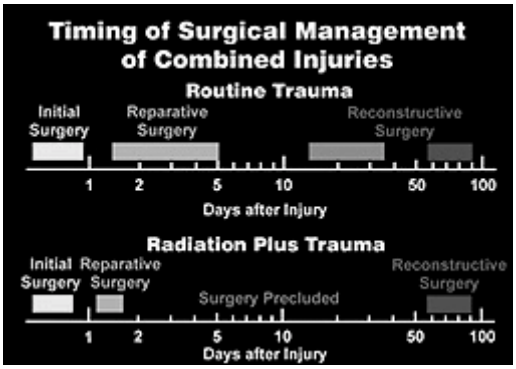

Figure 6-3. Timing of surgical management of combined injuries.

(1) If surgery or wounding occurs at any time other than during the prodromal symptom period, mortality is significantly enhanced. Low mortality wounds that either occur in the latent period, during manifest illness, or even during the apparent recovery period, prove excessively dangerous with at least a trebling of mortality. Early data was extrapolated into probability of patient survival, based on timing of surgeries. As surgery is extremely traumatic, any surgery that takes place after 48 hours will result in markedly increased mortality and morbidity. There is no data for the experimental models that receive aggressive hematopoietic resuscitation. Consequently, it is recommended that all surgeries be completed in the initial two days and that no further surgery, other than lifesaving interventions, occur until two months post injury.

(2) This dictum dramatically changes air evacuation priorities. Any patient who has a need for surgery must have definitive care surgical procedures completed within this short window. Small unit forward medical planning may violate this requirement as forward surgical teams (FSTs) will be required to perform not only lifesaving care, but also single surgery definitive care, with later revisions and repeat surgeries delayed at least 60 days.

6-28. Orthopedics

Early research with rabbit long bones demonstrates lack of adequate callus formation and subsequent nonunion in the irradiated animal. That is, animals that receive no treatment for irradiation will have nonunion of fractures. There has been no research into modern techniques of orthopedics and wound healing in the irradiated patient. At present, it is recommended that any reconstructive surgery be delayed until complete healing of the irradiation injury has occurred. There has also been no documentation of the effects of aggressive medical resuscitation in these patients. Primary amputation may be the most efficacious method of dealing with severely injured extremities. Conservative attempts at salvage by repeated debridement and reconstruction may well result in disaster for the irradiated patient.

6-29. Enteric Feeding

At the present time, it is believed that enteric feeding may be the best alternative even for those patients with radiological enteric mucosal damage (see Figure 6-4). The direct stimulation by nutrient drips appears to stimulate mucosal crypt formation. This regeneration of the damaged mucosal barriers inhibits bacterial movement from the lumen into the interstitial spaces. There is very limited research into this treatment regimen in the irradiated casualty, however, in nonirradiated trauma patients, TPN is inferior to direct enteric feedings. These data have not been replicated in trauma combined with radiation injury

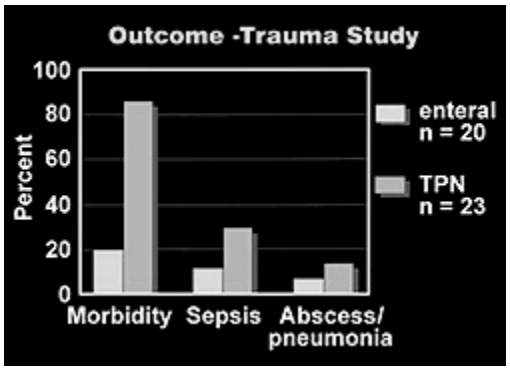

Figure 6-4. Enteric versus total parenteral nutrution.

6-30. Chemical Weapons and Radiation

Mustard agents and radiation can cause many similar effects at the cellular level. Their use in combination will have a geometric effect on morbidity. Research into these effects is only now just beginning. The immediate effects of the chemical agents must be countered before attention is paid to the effects of radiation that may not manifest for days or weeks. Little is known about the combined effect of radiation and nerve agents. Radiation will lower the threshold for seizure activity and may potentiate the effects on the CNS.

6-31. Biological Weapons and Radiation

The primary mechanism of death in radiation injury is infection by normal pathogens during the phase of manifest illness. Even minimally symptomatic doses of radiation depress the immune response and will dramatically increase the infectivity and apparent virulence of biological agents. Biological weapons may be significantly more devastating against a population that has been irradiated. Early research with radiation injury and anthrax simulant demonstrates that significantly fewer spores are required to induce infection. Computer simulations using these parameter changes yield orders of magnitude increases in casualties. Usually ineffective portals of infection that are made accessible by partial immunoincompetence may cause unusual infection profiles. Immunization efficacy will be diminished if instituted prior to complete immune system recovery. There are currently insufficient data to reliably predict casualties from combined injuries of subclinical or sublethal doses of ionizing radiation and exposure to aerosols with BW agent. Research suggests a shortened fatal course of disease when a virulent strain virus is injected into sublethally irradiated test models.

6-32. Immunization and Radiation

Previous immunizations may provide little or no protection to casualties with significant radiological injury. However, the secondary response of the irradiated immune system to previously recognized antigens has not been evaluated. Passive immunization against tetanus may be indicated in the presence of tetanus-prone injuries despite a nominally adequate prior immunization status. Use of live agent vaccines after irradiation injury could conceivably result in disseminated infection with the inoculation strain. No data is available on this phenomenon, but experience with immunocompromised patients predicts its occurrence. Preliminary investigations with nonvirulent agents and radiation injury indicate a significant level of infection will occur. Inoculation with live virus vaccines should be postponed until after complete recovery of the immune system. Killed virus vaccines may likewise fail to elicit an adequate immunogenic response. No data is available concerning cell-mediated immunity.

6-33. Nuclear Warfare Treatment Briefs

One of the products from the Medical Readiness Strategic Plan, fully supported by joint doctrine, involves the identification of joint NW/BW/CW patient conditions and the associated treatment protocols for inclusion into the Deployable Medical System database. The NW panel convened in November 1999 to focus on three injury categories likely to be encountered by military personnel with radiation injuries (radiation injury, trauma and burns). Twenty-two patient condition codes were identified and treatment briefs were developed that are applied at Levels I, II, and III MTFs. The proposed Nuclear Warfare Treatment Briefs are discussed in detail in Appendix B.

Section VI. EXTERNAL CONTAMINATION CASUALTIES

6-34. Internal/External Contamination

In addition to contamination resulting from the use of nuclear weapons, the probability of external and internal radioactive contamination has increased with the use of radionuclides in research projects, medical applications, nuclear power generation, and industrial processes. Among the hundreds of recorded accidents with significant radioactive contamination were those in Chernobyl in 1986 and Goiana in 1985. Each of the accidents resulted in many victims who required internal and external decontamination. Management strategies require knowledge of the radionuclides' physical and chemical characteristics, their metabolism in humans, and the methods of increasing their elimination from the body. Treatment should be a team effort that includes physicians, health physicists, analytical chemists, and toxicologists. External contamination is covered in this section.

6-35. External Contamination and Treatment

a. As noted in Chapter 4, significant amounts of radioactive material may be deposited on personnel and ground surfaces after the use of nuclear weapons and RDD; the destruction of nuclear reactors; nuclear accidents; or improper radiological waste disposal. Military operations in these contaminated areas could result in military personnel receiving sufficient radiation exposure or particulate contamination to warrant medical evaluation and remediation. There is also a significant risk of high explosive detonations and/or fire at a nuclear weapons accident site, especially in vehicular or aircraft accidents where oil or fuel is present.

b. Contamination of injured personnel may be due to either radioactive or toxic materials or both. In general, the hazard to both the patient and attending medical personnel will be so negligible that NECESSARY MEDICAL OR SURGICAL TREATMENT MUST NOT BE DELAYED BECAUSE OF POSSIBLE CONTAMINATION. That is, medical personnel should attend to life-threatening injuries and conditions first. Then, evaluate skin and clothing contamination. If external contamination is detected, internal contamination is highly likely. The initial evaluation for patient contamination includes an operational history of the patient’s unit, or a background of the accident, initial area and patient surveys, and nasal swipes of the patient. Decontamination should be done as soon as possible during the care of such patients, and ideally, prior to admission to an MTF. Decontamination procedures should be part of the operational plans and guides of all medical units and departments of fixed medical facilities. This ensures flexibility of response and action and will prevent delay in needed medical treatment. The simple removal of outer clothing and shoes will, in most instances, affect a 90 to 95 percent reduction in the patient's contamination.

c. Medical personnel minimize the risk of exposure by following decontamination principles similar to those for BW and CW agents. If circumstances allow, medical personnel should don protective clothing before coming into contact with contamination. Protective clothing consists of gloves, overshoes, and a plastic apron. Surgical gowns are acceptable.

Contain irrigation fluid in holding tanks and bag contaminated clothing and medical supplies and give them to radiation safety personnel. A maximum limit of 0.05 cGy has been established for US medical personnel who provide care for radiation victims. The highest actual dose recorded for a US health care worker was 0.014 cGy, which occurred during the care of a radiation accident victim of a commercial nuclear power plant accident. That dose approximates the dose received during a single chest radiograph.

d. Use beta-gamma and alpha monitoring instruments for the initial radiation survey of the skin and clothing. If contamination is present on the clothing, remove it and repeat the monitoring over the patient's skin. Contaminants may be held to the surface of the skin by electrostatic forces, surface tension, or binding with skin proteins. Depth of the skin is important relative to type of radiation. Alpha rays on the skin surface do not reach the basal cell layer of the epidermis. Beta rays are reduced by a factor of two for every 1 mm of skin. Skin on most areas of the body has a depth of 2 mm. The epidermis is approximately 0.1 mm in depth, except over areas of external friction. Those areas include the palms, digits, and soles of the feet where the thickness of the stratum corneum can reach 1.4 mm. Health physicists use the estimate of skin radiation dose at the basal epithelium, since that is the area that lies adjacent to the small blood vessels of the dermis. This is the area that can be affected by beta and gamma rays.

(1) *Skin Decontamination*. Skin decontamination should be undertaken to decrease the risk of skin beta burns, to lower the risk of internal contamination of the patient, and to reduce the potential of contaminating medical personnel and the environment. After the patient's clothing is removed, washing the patient with soap and water is 95 percent effective because soap emulsifies and dissolves contamination. Gentle brushing or the use of an abrasive soap or abrasive granules dislodge some contamination physically held by skin protein, or removes a portion of the horny layer of the skin. Addition of a chelating agent helps by binding the contaminant in a complex as it is freed from the skin. Keep in mind that the stratum corneum of the epithelium is replaced every 12 to 15 days. Thus, contamination that is not removed and is not absorbed by the body will be sloughed within a few days.

(2) *Decontamination Techniques*. Avoid unnecessary damage to the skin; cease washing before abrasion occurs. A detector count of less than 1 mrem per hour of beta and less than 1,000 disintegration's per minute of alpha are safe decontamination end points. For stubborn hand and distal extremity skin contamination that is not removed by washing, wrap the contaminated area and, over time, sweating will decrease contamination. To decontaminate hair, use any commercial shampoo without conditioner. Conditioners bind material to hair protein, making contamination removal more difficult. Consider clipping or shaving hair to remove alpha contaminants. Do not remove eyebrows without significant cause since they grow back slowly if at all. For skin and wound decontamination, use a cleaning solution. Suggested solutions are--

- Soap and water or normal saline.
- Betadine and water.
- Phisoderm and water.

- Hydrogen peroxide.
- Dakin solution (0.25 percent sodium hypochlorite).

Section VII. INTERNAL CONTAMINATION AND TREATMENT

6-36. Internal Contamination Sources and Hazards

a. In a nuclear explosion, more than 400 radioactive isotopes are released into the biosphere, of which about 40 are potentially hazardous to humans. This fallout may be deposited onto clothing and/or skin and, then, may enter the body. Gaseous material or particulate matter may be inhaled and subsequently absorbed or deposited throughout the respiratory tract. Radioactive material that falls onto food or into the water supply or that is transferred from hand to mouth may be ingested. A source of chronic exposure is radioactive material incorporated into the food chain, as in the case of contaminated cow's milk and mushrooms in countries of the former Soviet Union after the Chernobyl accident. Still other sources of contamination are medical misadministrations and the release into the environment of medical radioactive materials. Examples include the injection of soluble phosphorus-32 instead of insoluble phosphorus-32 and overdosing with iodine-131 by confusing microgram with milligram. For a detailed discussion of potential contamination sources, see Chapter 2, Section II.

b. The extent of a contamination hazard is determined by a number of factors:

- Amount of radionuclide(s).
- Energy and type of radiation.
- Duration of external and/or internal exposure.
- Effect on critical organs. Some internalized chemicals, including the radionuclide sodium-25, are evenly distributed throughout the human body. Others have an affinity for specific organs (target organs) where the radionuclides concentrate due to their particular chemical properties and metabolic pathways. For example, lead's critical organ is bone. Although lead is initially distributed to soft tissue organs, including the kidneys, liver, and RBCs, it is redistributed to bone where it forms long-lasting bonds, and then it is slowly eliminated. Iodine's critical organ is the thyroid gland, and uranium's critical organs are the kidneys, liver, and bone.
- Chemical makeup of the radionuclide. Heavy metals, like uranium, are renal and hepatic toxins; radioactive isotopes of uranium compounds this toxicity.

6-37. Internal Contamination Mechanisms

Fine particles from nuclear fallout and smoke from fires of a nuclear accident will contain a large variety of material from the weapons themselves, the transport vehicle, and the environment. Some of these materials can be dangerous if inhaled. Particles smaller than about 10 microns in the smoke from a fire, if inhaled, may penetrate deeply into the respiratory system where the probability of retention is high. This can result in significant damage to the lungs. Early recognition of internal contamination provides the greatest opportunity for contaminant removal and reduces the potential for injury. Radionuclides obey the same principles of toxicity as do nonradioactive toxins. Basically, toxins are absorbed into the body and then distributed throughout the body or, for some chemicals, concentrated in critical organs. A toxicant may undergo metabolic changes in the liver or in target organs and become more active or less active metabolites. Ultimately, the toxicant is eliminated through the body's excretory mechanisms. The internalization process consists of intake, distribution, and metabolism phases.

a. *Intake.* In order of decreasing frequency, contaminants enter the body by the following four principle intake routes:

- Inhalation.
- Ingestion.
- Wound contamination.
- Absorption.

(1) *Inhalation*. The inhalation pathway is the primary intake route for radioactive contamination. Absorption is dependent on solubility for liquid and solid contaminants. The contaminant's particle size determines its deposition within the respiratory tract. For example, particles smaller than 25 microns in diameter may reach the alveolar area. Particulates greater than 25 microns are too large to pass into the alveoli and are deposited in the upper airways. After deposition, absorption depends on the chemical solubility of the contaminant. Soluble particles are absorbed directly into the blood stream, or they pass through the lymphatic system and, ultimately, move into the circulatory system. The mucociliary apparatus clears insoluble particles deposited above the alveolar level within the respiratory tract. Most secretions that reach the pharynx are swallowed and enter the GI system. Insoluble particles irradiate surrounding tissues until the particles are cleared from the respiratory tract (see Table 6-16). Fibrosis and scarring are likely to occur in the alveoli due to the localized inflammatory response to foreign bodies.

Table 6-16. Clearance Times from the Respiratory Tract

Structure	Clearance Time (hours)	Cumulative Time (hours)
Trachea	0.1	0.1
Bronchi	1.0	1.1
Bronchioles	4.0	5.1

Terminal Bronchioles	10.0	15.1
Alveoli	100+ days	100+ days

(2) *Ingestion*. The ingestion pathway consists of the GI tract. Absorption of the radionuclide depends on its chemical characteristics and on its solubility. For example, the absorption rate of radium is 20 percent and strontium is 30 percent. However, 100 percent of tritium, iodine, and cesium are absorbed. The GI tract is the critical organ for ingested, insoluble radionuclides. The large intestine receives the greatest radiation exposure due to its slower transit time. Insoluble alpha particles cause no significant injury, even in individuals with low-fiber diets and slow transit times, because the exposure time within the critical organ is relatively short (see Table 6-17).

Table 6-17, Clearance Times of the Human Gastrointestinal Tract

Organ	**Mean Emptying Time (hours)**	**Average Occupancy Time (hours per day)**
Stomach	1	6
Small Intestine	4	14
Upper Large Intestine	13-20	18
Lower Large Intestine	24	22

(3) *Wound contamination.* Wounds are classified as abrasions, lacerations, or punctures. The differing characteristics of each type of wound affect the absorption and decontamination of radioactive substances. Abrasions present a large surface area denuded of intact skin; this decreases the skin barrier and increases the potential for absorption. Lacerations usually offer easy access for decontamination by cleaning or removal of contaminated tissue by excision. Puncture wounds offer minimal access for decontamination, and they make it difficult to determine the wound depth and the deposition of contamination. Solubility, pH, tissue reactivity, and particle size determine the speed of contaminant absorption within a wound. The more soluble the contaminant, the greater the absorption rate. Comparatively small particles may phagocytize and enter the lymphatic system. If the contaminant is highly acidic, local tissue coagulates, decreasing the dispersion rate. For example, human wounds that contain depleted uranium may develop cystic lesions that decrease the uranium absorption rate. This has been demonstrated in Gulf War veterans wounded by DU shrapnel. Studies in these veterans and in animal models have demonstrated that uranium from these wounds is slowly distributed to the liver and kidneys (see Section IX).

(4) *Absorption.* The skin acts as a physical barrier with the horny epithelial layer acting as the primary barrier. Percutaneous absorption occurs by passive diffusion. Every substance has a skin permeability coefficient that depends on the substance's solubility. Skin that has been mechanically damaged, as from repeated abrasive scrubbing, allows for greater absorption. Skin that has been exposed to certain chemicals like dimethyl sulfoxide is also more permeable. Absorption through sweat glands and hair follicles is a minor concern since, overall, they constitute only a small surface area.

b. Distribution. Once a radionuclide is absorbed, it crosses capillary membranes, through passive and active diffusion mechanisms, and then is distributed throughout the body. The rate of distribution to each organ is relative to the blood flow through the organ, the ease of chemical transport across cell barriers, and the affinity of the radionuclide for naturally occurring chemicals within the organ. The liver, kidney, adipose tissue, and bone have relatively high capacities for binding chemicals because they are high in proteins and lipids.

c. Metabolism. After absorption, distribution, and uptake, a radionuclide is excreted either in its original state or as a metabolite. A radionuclide is metabolized according to its chemical properties and the principal organ for metabolism is the liver. The biologic half-life of a radionuclide is as important as its radiological half-life in determining the significance of the exposure. The primary route of excretion is through the urinary tract, followed by excretion through the liver and lungs. Minor routes of excretion include sweat, saliva, milk, and seminal fluid. The excretion route is determined by the relative solubility of the compound and its delivery to the appropriate organ. In general, compounds that are water soluble are excreted through the urine, while lipid-soluble compounds are secreted via the bile into the intestine (see Figure 6-5).

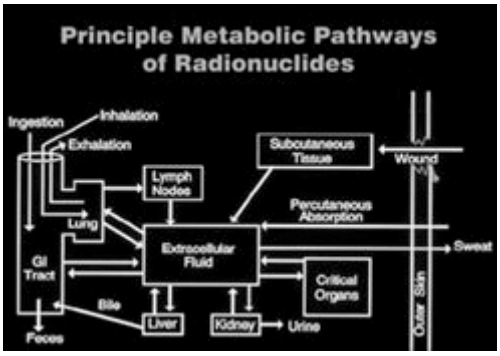

Figure 6-5. Metabolic pathways.

6-38. Internal Contamination Treatment

Once a contaminant is absorbed, inhaled, or ingested, it is important to decrease the uptake into the circulatory system, to decrease the deposition in critical organs, and to increase the excretory rate of the contaminant. A number of procedures are available for respiratory contamination and GI contamination. As with any medical treatment, the clinician should consider the risks and benefits to the patient. The benefit of removing the radioactive contaminant with modalities associated with significant side effects and morbidity must be weighed against the short- and long-term effects of contamination without treatment. The radioactivity and the toxicity of the chemical must also be considered. Risk estimates include the professional judgement combined with the statistical probability of radiation-induced disease occurring within a patient's lifetime. Some of the treatment agents which could be used are discussed below.

a. Prussian Blue. The US Food and Drug Administration has removed earlier restrictions on the use of Prussian Blue (ferric ferrocyanide) in the US. Prussian blue is indicated

for cesium, thallium, and rubidium contamination. Other investigational drugs are also authorized for use under approved study conditions. This chemical is not absorbed by the GI tract and works through two modes of action. It decreases the absorption of many radionuclides into the GI tract and removes some radionuclides from the capillary bed surrounding the intestine and prevents their reabsorption. Prussian Blue is most effective when given early after ingestion and serially thereafter. In animals, Prussian Blue is effective in removing cesium, thallium, and rubidium by the fecal route. It has been used, with serious side effects, outside the US to treat humans for cesium-137 internal contamination. In Brazil, following the release of cesium-137 from a medical radiation source, Prussian Blue was used to treat contaminated human victims. Cesium-137 is easily absorbed by the body through ingestion, inhalation, and skin penetration. Its physical half-life is 30 years, and its biologic half-life is 100 days. It emits beta and penetrating gamma radiation and is distributed uniformly throughout the body. Prussian Blue decreases the biological half-life of cesium-137 to 30 percent of the original 100 days. Also, *in vitro* studies demonstrated that cesium-137 can be removed from dialysis fluid by using columns coated with Prussian Blue. Prussian Blue is approved as an investigational new drug, and the Radiation Emergency Assistance Center/Training Site has the license for research use.

b. Blocking and Diluting Agents. Blocking and diluting agents work by preventing the uptake of a radionuclide in a critical organ or by overwhelming the critical organ with stable compounds that reduce the uptake and incorporation of the radionuclide. Potassium iodide is an excellent example of a blocking agent. If given within 30 minutes of exposure to iodine-131, potassium iodide prevents the uptake of iodine-131 by the thyroid gland. If given within the first 6 hours, the blocking effectiveness is 50 percent. At 24 hours, its blocking effect is zero. Potassium iodide should be given for 7 to 14 days to prevent the uptake of recycled iodine-131. The recommended daily dose of iodide is 300 milligrams given as 390 milligrams of potassium iodide. Any readily available soluble form of iodine with equivalent iodide content is suitable. Some cough syrups contain sufficient iodide to achieve an effective dose. Enteric-coated tablets should be crushed to allow for more rapid absorption. Toxic side effects from potassium iodide are rare, though some highly sensitive individuals develop angioedema. Chronic iodide administration may cause acute toxicity with rhinitis, conjunctivitis, headache, drug fever, and skin rashes; however, withdrawal of the drug results in recovery. Potassium perchlorate (200 milligrams by mouth daily) may be given to individuals who are sensitive to iodine. Stable strontium in the form of strontium lactate, given by mouth in doses of 500 to 1,500 milligrams daily for several weeks decreases strontium-85 and strontium-90 uptake in bone and testes. Water (three to four liters peritoneally) increases tritium urine concentration.

c. Mobilizing agents.

(1) Mobilizing agents are compounds that increase the metabolic rate of internal contaminants, resulting in an increase in elimination. Examples of mobilizing agents are the antithyroid medications--propylthiouracil, methimazole, and potassium thiocyanate. In theory, use of a medication that reduces the manufacture of thyroid hormone (T3 and T4), also reduces the presence of radioactive iodine in this critical organ. Once the thyroid has manufactured thyroid hormone, the half-life of the hormone is 120 days. Iodine, which has not been oxidized and incorporated into thyroid hormone, is excreted at a much faster rate. Propylthiouracil and methimazole block the oxidation of the iodide ion required in the synthesis

of thyroid hormone, thus reducing the production of thyroid hormone. Propylthiuracil and methimazole effects are short acting, and these drugs must be given every 8 hours to be effective. If the thyroid has an ample supply of iodide, these drugs are less effective. Potassium thiocyanate reduces the iodide gradient that exists between the thyroid and the blood. Normally the gradient is 50 to 1. A single dose of thiocyanate reduces the iodide-concentrating capacity of the thyroid gland and increases the excretion of stored iodide from the thyroid. The toxicity of these three antithyroid drugs and the relative ineffectiveness make them less appealing for use then potassium iodide.

(2) Ammonium chloride, an acidifying salt given orally, mobilizes strontium from body tissues and, if given with calcium gluconate intravenously, causes a 40 percent to 75 percent decrease in body stores of strontium over a period of 3 to 6 days. The combined treatment is most effective if given early after strontium deposition, but some effectiveness is still demonstrated if given as late as 2 weeks after deposition. Side effects include gastric irritation, nausea, vomiting, and hepatic toxicity in some individuals.

(3) Diuretics are known to decrease sodium, potassium, and chloride serum levels. In theory, but not proven in humans, diuretics may be useful in reducing sodium-22, sodium-24, potassium-42, and tritium levels.

d. Chelation agents. Chelators are mobilizing agents; they enhance the elimination of metals from critical organs. Chelators are organic compounds (ligands) that exchange less firmly bonded ions for metal ions. The stable complex, the chelator and the metal, is then excreted by the kidney. Chelation therapy has been used for lead, mercury, arsenic, and other heavy metals. In addition, chelation therapy has been used to treat internal radiation contamination on a limited basis. Chelators are most effective prior to binding of a toxicant in the critical organ but can be used with variable results after target organ acquisition. Before, during, and after chelation therapy, pertinent radioactivity measurements should be made of urine and feces, and total-body and chest counting should be performed. The most effective dose schedules have not been determined. As with most chelators, it is more effective the earlier it is given. Both salts can be given IV or as a nasal inhalant. Dose recommendations are, for adults, 1 gram in 100 to 250 cc of normal saline infused IV over 3 to 4 minutes and repeated on 5 successive days per week. Given through the aerosolized route, 1 gram in a 4 cc vial is placed in a nebulizer, and the entire volume is inhaled over 3 to 4 minutes and repeated daily. Also, before, during, and after chelation therapy, pertinent measurements for radioactivity should be made to determine the efficacy of treatment. By the fifth day, evaluate the bioassay data of urine and feces samples and the total-body and chest count measurements. Continuation of therapy is determined by assessing chelation yield with remaining body burden. No serious toxicity in humans has been reported when used in recommended doses. When given repeatedly with short intervals for recovery, nausea, vomiting, diarrhea, chills, fever, pruritus, and muscle cramps have been noted.

(1) New chelators, including DMSA (meso-2,3,-dimercaptosurrinic acid) for lead toxicity and DMPS (2,3 dimercapto-1-propanesulfonic acid) for mercury toxicity, have not been used for metals, including plutonium and americium. Sodium bicarbonate has been used to treat uranium contamination. At this writing, investigations are looking at methods to increase

the mobilization of uranium from wounds. The compound 3,4,4 LIHOPO (a hydroxypyridizone ligand), if given immediately after wound contamination in rats, mobilizes the uranium and allows for excretion in the urine.

(2) One of the older chelators, CaEDTA (calcium ethylenediaminetetraacetate), was used extensively in the past to treat lead intoxication, and it has been used to treat plutonium and americium toxicity. Given intravenously or intramuscularly, it is painful and has significant side effects, including GI upset, pain at the injection site, bone marrow depression, and nephrotoxicity.

(3) The chelator DTPA (diethylenetriaminepentaacetic acid), in the zinc or calcium salt state, forms stable soluble complexes with a large number of metal ions. When DTPA releases its calcium or zinc, it binds to soluble plutonium, americium, or curium and carries it to the kidneys where it is then excreted in the urine. The plasma half-life of CaDTPA is 20 to 60 minutes. No accumulation of DTPA occurs in tissues or specific organs. CaDTPA is approximately ten times more effective than ZnDTPA (zinc DTPA) for initial chelation of transuranics. Therefore, CaDTPA should be used whenever larger body burdens of transuranics are involved. Diethylenetriaminepentaacetic acid has been shown to greatly reduce the uptake of absorbed P-239 if given within an hour of contamination. With repeated dosing, CaDTPA can deplete the body of zinc and, to a lesser extent, manganese. Zinc replacement therapy is recommended when repeated dosing is done due to loss of the body's zinc stores. Contraindications for CaDTPA are minors, pregnant women, nephrotics, and persons with bone marrow depression. Teratogenicity and fetal death have occurred in mice.

6-39. Contamination Measurement

A number of methods are used to detect contamination and to estimate the extent of contamination. Direct methods measure skin and body contamination with hand-held and stationary radiac instruments. Indirect methods detect and quantify radioactivity in excretory products; this measurement is used with extrapolation curves to estimate body burden.

a. Direct Contamination Measurement. Direct measurement methods include total-body and partial-body counters that also detect skin and wound contamination. The advantage of direct measurement is that it does not require the use of excretory rates for estimation of contamination. A major disadvantage is that measurements are influenced by external contamination and background radiation levels. Total- and partial-body counters measure beta and gamma radiations that are powerful enough to reach the body surface. Partial-body counters are used for chest and thyroid measurements. Chest counters detect respiratory tract levels of contaminants such as plutonium and uranium. Surface detectors are usually used for skin and wound monitoring in the field. Small probes may be used for deep wounds and can be cold sterilized for this purpose. Contaminated wounds with alpha particles are difficult to detect because blood or body issue may block the radiation. Therefore, alpha contamination measurement usually relies on detection of the gamma/beta radioactivity of daughter products or other contaminants.

b. Indirect Contamination Measurement.

(1) Skin swipes and nasal swipes are used to estimate the extent and type of contamination that has been internalized. Nasal swipes are taken bilaterally, using moistened, cotton-tipped applicators to swab the nares. The swabs are then placed individually in test tubes or envelopes, which are labeled with the subject's name and the sample collection time and date. The swipes are sent to a facility with a laboratory counter where contamination can be measured. The detection of radioactive material in the nares indicates respiratory inhalation. Unilateral contamination may indicate only surface contamination.

(2) Bioassay sampling of urine and feces also provides indirect measurement of body deposition. Radioactivity and concentration of the nuclide in urine and feces depend on individual metabolic and clearance rates. Extrapolation curves to estimate internal contamination are based on average human metabolic and clearance rates. Bioassay sampling and excretion data are the principal methods of determining the presence of alpha and pure beta emitters, which are the most hazardous internal contaminants. Initial samples to be used to establish baseline levels of urine and fecal radioactivity should be obtained from a patient as soon as the medical condition allows. Measures should be taken to avoid accidental contamination of samples. For example, contaminated clothing from the victim should be removed and initial skin decontamination steps should be accomplished before sampling, and gloves should be worn by all personnel handling capture containers. Bioassay accuracy depends on baseline levels, multiple postexposure samples, and knowledge of the precise time of contamination and of the type of contaminant(s) (see Table 6-18).

Table 6-18. Guidelines for Bioassay Sampling

Optimum Sample Time After Exposure			
Material	**Feces**	**Urine**	**Quantity**
Plutonium	24 hours	2-3 weeks	24 hour total
Uranium	24 hours	24 hours	24 hour total
Tritium	N/A	12 hours	1 voiding

Section VIII. DELAYED/LATE EFFECTS

6-40. General

a. Delayed or late effects of radiation occur following a wide range of doses and dose rates. Delayed effects may appear months to years after irradiation and include a wide variety of effects involving almost all tissues or organs. Radiation exposure contributes to threat hazards by the risk of inducing latent injuries. Some of the possible delayed consequences of radiation injury are carcinogenesis, cataract formation, chronic radiodermatitis, decreased fertility, and genetic mutations. However, it should be emphasized that the Hiroshima, Nagasaki,

and Russian experiences have not shown significant genetic effects in humans, although exposure to ionizing radiation at levels exceeding background carries an adverse health risk. At the lower levels of exposure (background levels to 0.7 Gy), this *risk* tends to be probabilistic in nature, relating more to exposed populations than to exposed individuals. Health risks incurred tend to be long-term in nature, and not immediate, therefore lacking significant operational impact. These risks may, however, manifest themselves as a significant disease long after the operation.

b. In relation to associated long-term health risks, several principles need to be reviewed. First, exposure to ionizing radiation that exceeds background levels carries a risk to develop deleterious long-term health effects. Second, the health risk incurred by the exposure is generally related, and often times, proportional to dose. In addition, biological factors relative to the irradiated individual need to be considered; for example, age and sex of the individual, health status, and the individual's genetic makeup. In addition to the total dose factor, radiological parameters that factor into long-term health risks include--

- Exposure rate and quality of the radiation.
- Location of the source (external versus internal).
- Nature of exposure (continuous versus fractionated versus protracted; prompt external exposure versus chronic committed dosing).
- Time after exposure and requisite repair times and latency times required for pathologies to manifest.

c. Deterministic effects are those that are directly dose related. They include both acute and delayed effects. The greater the radiation exposure, the greater the magnitude of the effect. While individual variations will occur due to individual sensitivity, the intensity of the effect is still directly dose related. Tissue fibrosis, chronic immune system suppression, reproductive tissue dysfunction, and selected ocular problems are some of the more common (and serious) symptoms of the late-arising deterministic pathologies. Formation of ocular cataracts is the most common delayed radiation injury. Higher doses tend to increase the degree of opacity and shorten the period of latency. There is a 10 percent chance of developing a severely impairing cataract following a single exposure to 2.4 Gy of low LET radiation, and a 50 percent incidence following a dose of 3.1 Gy. Immune system defects occur at doses of 0.5 Gy and larger.

d. A stochastic effect is a consequence based on statistical probability. For radiation, tumor induction is the most important long-term sequelae for a dose of less than 1 Sv. Most of the data utilized to construct risk estimates are taken from radiation doses greater than 1 Sv, and then extrapolated down for low-dose probability estimates. Significant direct data are not available for absolute risk determination of doses less than 50 mSv.

(1) Scientific consensus reports have estimated that the life-time risk of fatal cancer occurrence is increased by 770 cases per 100,000 persons/0.1 Sv for males and 810 cases

per 100,000 persons/0.1 Sv for females. To illustrate this effect, the US background fatal cancer incidence rate is 20,000 cases per 100,000 persons. Therefore, if a mixed group of 100,000 people receive 0.1 Sv single dose irradiation, instead of 20,000 cancers, approximately 20,800 fatal cancers would occur. Deciding which 800 of these 20,800 cases were radiation-induced would be difficult. National cancer incidence rates do vary as do the corresponding risk estimates and account should be taken of these whenever possible.

(2) Genetic abnormalities are also stochastic, as their occurrence is based on genetic material damage that is minor enough to allow cell replication, but too severe to be repaired by normal protease action, that is, DNA double-strand breaks. Studies estimate that an effective dose of 1 Sv would double the incidence of congenital abnormalities. The current background incidence rate of occurrence is 20 to 30 thousand per million live births.

6-41. Pathologies of Concern

Late-arising consequences of a prior exposure encompass not only somatic effects, but also genetic and teratogenic effects as well. However, only selected classes of somatic responses are sufficiently well documented in man as to merit a high degree of concern for military operations.

a. Significant long-term genetic effects remain at best ill-defined and unconfirmed. (Although they are suggested by selected epidemiological studies that purport an elevated cancer incidence in children of nuclear workers.) Further, there have been recent reports of radiation induced stable genetic changes (somatic mutations) being detected by sophisticated molecular assays of individuals exposed at Chernobyl. Although interesting, the significance of these changes in terms of long-term health remains unclear.

b. Radiation-induced teratogenic effects, by contrast, have been clearly documented by the increased mental retardation in Japanese children irradiated *in utero* as result of the nuclear bomb detonations over Hiroshima and Nagasaki. The direct military relevance of this teratogenic effect (as well as related ones, including microcephaly, microphthamia, reduced growth, skeletal defects, neoplasias, and cataracts) is questionable. Further, the teratogenic responses appear to have a broad exposure threshold for induction, with significant responses being noted only at doses greater than 0.5 Gy.

6-42. Carcinogenesis

Irradiation of almost any part of the body increases the probability of cancer. The type formed depends on such factors as area irradiated, radiation dose, age, and other demographic factors. Irradiation may either increase the absolute incidence of cancer or accelerate the time or onset of cancer appearance, or both. There is a latent period between the exposure and the clinical appearance of the cancer. In the case of the various radiation-induced cancers seen in man, the latency period may be several years. Latent periods for induction of skin cancers in man have ranged from 10 to 50 years after therapeutic X-ray exposures, to a reported 15 years for bone

tumors after radium exposure. This latency related to bone tumors is very dependent upon the dose and type of radiation emitted by the radionuclide.

a. A leukemogenic effect was expected and found among Hiroshima and Nagasaki survivors. The peak incidence occurred 6 years after exposure and was less marked for chronic granulocytic leukemia than for acute leukemia. British men receiving radiotherapy for spondylitis showed a dose response relationship for leukemia, with peak incidence occurring 5 years after the first exposure. Studies have demonstrated that ionizing radiation can induce more than one kind of leukemia in man, but not chronic lymphocytic leukemia.

b. It is difficult to address the radiation-induced cancer risk of an individual patient due to the already high background risk of developing cancer. The more important radiobiological conditions that factor into cancer induction (or for that matter any of the somatic effects) include those parameters previously mentioned, namely dose, dose-rate, and radiation quality. Cancer is not a single disease, but a complex of diseases comprised of both cancers of the blood (leukemias), and cancers of solid tissues of both epithelial and mesothelial origins. The radiogenic nature of these specific cancers differs substantially. Bone tumors (osteosarcomas) serve as a good example, as they are prominent late arising pathologies associated with internally deposited, bone-seeking radionuclides (^{90}Sr), but are rarely associated with the cancers that stem from exposure to external radiation sources (^{60}Co).

c. Cancer types that are unequivocally inducible by ionizing radiation are the lymphohematopoietic cancers, cancers of the lung, mammary tissues, liver, thyroid, colon, stomach, pancreas, salivary glands, and kidneys. Cancers with either a low incidence or a low probability of induction include cancers of the larynx, nasal sinuses, parathyroid, nervous tissue, and connective tissue. Cancers that are probably not inducible include the chronic lymphocyte leukemias and cancers of the uterus, cervix, prostate, testis, mesentry, and mesothelium.

d. Cancer occurrence following radiation exposure is probabilistic in nature, based more on group responses and less on individual responses, per se. The uncertainly of the dose-response relationship is indeed significant and has led to a number of different models being proposed. None of the models have been proven truly satisfactory in low-dose ranges. One of the more conservative models is the *linear, no-threshold* model. This model postulates that all dose-rates and absorbed doses of radiation can induce cancer, and that the slope of the line remains essentially constant. Despite the *convenience* of using this model in a generic way to assess cancer risk, specific cancer types exhibit different dose-response relationships. Leukemias, for example, show a shallow sloped initial linear response that extends into a quadratic function. Solid tumors, by contrast, tend to show a more steep sloped linear response over the entire exposure range. Based on linear, no-threshold modeling of dose-relationships of the Japanese survivor data, the overall frequency of cancer (lifetime risks) following prompt, low LET, whole body irradiation is estimated to be approximately 7 percent per Gy. Under protracted exposures, the frequency decreases to about one half this value, or about 3-1/2 percent per Gy. Using the assumptions of linear, no-threshold modeling, the lifetime rates of fatal cancer induction in acute and protracted exposures (upper-end values) for the five subcategories of RES 1 unit exposures are listed in Table 6-19. Such dose dependent cancer frequencies should at best

only be considered as rough estimates based on a series of assumptions, including the basic shape of the cancer induction curve below 0.5 Gy.

Table 6-19. Increased Cancer Incidence by Unit Radiation Exposure Status

Unit Radiation Exposure Status	1A	1B	1C	1D	1E
Acute Radiation	0.03%	0.35%	0.70%	1.8%	4.9%
Protracted Radiation	0.015%	0.18%	0.35%	0.90%	2.5%

*Linear, no-threshold model

e. The overall estimates for excess radiation-induced cancers in male and female personnel, ranging in age from 15 to 45 years, is ~0.8 percent and ~1.0 percent per 0.1 Sv, respectively. These radiogenic cancers of man can be grouped into four broad categories, with each carrying varying degrees of risk as follows:

- Cancers of the blood-forming system (leukemias). Induction frequencies of ~0.08 percent per 0.1 Sv for males and ~0.06 percent per Sv for females are estimated.

- Cancers of the respiratory tract (trachea, bronchus, and lung). Induction frequencies of ~0.2 percent are estimated for both sexes.

- Cancers of the digestive system (including stomach, colon, liver, and pancreas). Excess cancer rates are estimated to be in the range of 0.2 percent per Sv for males and 0.4 percent per Sv for females.

- Remaining cancers of various tissues and organ systems. The estimated incidence is about 0.3 percent per Sv for both males and females. For cancers of the breast, only females show excess to develop radiation-induced cancers, having an estimated frequency of 0.1 percent per Sv.

f. Another issue relates to the health hazards associated with internally deposited radionuclides. Significant health risks are associated with *internalized radionuclides*. These risks are largely long-term in nature and depend not only on the species and concentration of nuclide absorbed, but also on the nature of the exposed individual. Cancers of the lung, liver and bone are the principal areas of concern. Although large threshold doses are generally required to induce these cancers, once threshold exposure levels have been surpassed, high frequency of tumor development can occur (for example, ~6 percent bone sarcomas in radium exposed individuals).

6-43. Cataract Formation

A late effect of eye irradiation is cataract formation. It may begin anywhere from 6 months to several years after exposure. While all types of ionizing radiation may induce cataract formation, neutron irradiation is especially effective in its formation, even at relatively low doses. Cataract formation begins at the posterior pole of the lens and continues until the entire lens has been affected. Growth of the opacity may stop at any point. The rate of growth and the degree of opacity are dependent upon the dose as well as the type of radiation. The threshold for detectable cataract formation is 2 Gy for acute radiation doses and 15 Gy for protracted doses. A 50 percent cataract risk has been estimated at acute doses of approximately 3 Gy. This estimate assumes a low LET exposure, and it has been recently suggested that with high LET particle irradiation, the initiating cataractogenic dose might be considerably lower, well within 0.7 Gy.

6-44. Reproductive Tissue Disturbances

a. In males, sterility is induced at about a 50 percent incidence with an acute dose of approximately 0.7 Gy. Fractionated exposures fail to elicit a significant sparing effect; for instance, a 50 percent incidence of aspermia was noted at a 0.35 Gy dose given under a protracted exposure regimen. Temporary male sterility due to damage to spermatogonia will occur after 0.1 Gy of local or whole body irradiation. As this is a maturation depletion process, the azospermia will not occur until two months after irradiation. Protracted radiation exposure will cause a more prolonged episode of azospermia. Serum levels of testosterone will be unaffected.

b. Female reproductive tissues appear more resistant. Doses above 1.5 Gy impair female fertility and may cause permanent sterility, with older women more sensitive to the effects due to the presence of fewer oocytes. Induced sterility occurs at moderately high doses; it has been noted that 2.5 to 5 Gy induces permanent sterility in about 60 percent of irradiated women.

6-45. Cutaneous Effects

Acute radiation exposure hazards are commonly addressed with respect to total body gamma or neutron irradiation. Bone marrow failure consequently becomes the primary clinically relevant aspect of the acute radiation injury. In many cases of accidental exposure, high doses of radiation may be delivered to only a portion of the body (partial body exposure). Under these conditions of nonhomogeneous exposure, local organ systems, such as the skin, may become more important in determining clinical prognosis. The severity of the skin injury is disproportionate to the total body dose, consequently, the skin damage exceeds the manifestation of ARS. This was especially obvious in the Chernobyl and Goiânia accidents. The primary cause of cutaneous lesions in the Chernobyl survivors was short-range radiation from nuclides in cutaneous contamination. Only the upper parts of the dermis and subcutaneous tissue were affected. The larger vessels penetrating the muscle fascia were only minimally harmed.

a. Symptoms. Cutaneous radiation effects follow a distinct clinical pattern that defines the CRS. Within minutes to hours after exposure, an erythematous reaction develops that

may be associated with a *burning* urticaria. This transient prodromal stage usually lasts less than 36 hours. It is followed by a clinically inapparent latent stage. The manifest stage is characterized by occurrence of an intensively erythematous skin, which may show scaling and desquamation. In more severe conditions, subepidermal blisters and even ulcerations may develop. Though similar skin lesions are produced by thermal injury, the time course and underlying processes involved in the development of the CRS are so different from thermal burns that the terms *radiation burns* or *beta burns* are considered inappropriate and misleading for this clinical condition and should therefore be abandoned.

b. Treatment and Follow-up of the Cutaneous Radiation Syndrome. Standardization of treatment is difficult to achieve due to the rarity of true radiation accidents. An established treatment scheme, which would go beyond recommendations, based on anecdotal observations, does not exist. Differing procedures in documentation of accidents further reduce the intercomparability of therapeutic efforts in accident situations. Whatever the circumstance, treatment must provide symptomatic relief and minimization of additional risk to the patient. Recommended therapies, dosages, and the therapeutic outcome are summarized in Table 6-20.

Table 6-20. Symptom-Oriented Therapy for the Cutaneous Radiation Syndrome

Symptom	Treatment	Application	Dosage	Result	Side Effects
Pruritus	Anti-histamines	Oral	As appropriate	Relief of itch	Sedation
Erythema	Steroids	Topical.	2 X daily	Alleviation	None when used less than 3 weeks
Blisters	Steroids TCDO	Wet dressing	3 X daily.	Alleviation	
Dryness	Linoleic acid cream	Topical	1 X daily	Inhibition of water loss	
Keratoses	Tretinoin Acitretin	Topical oral	1 X daily 0.1-0.3 mg/kg	Clearance moderate	Irritation; dryness of lips
Inflammation	Mometasone	Topical	3-4 X week	Alleviation	
Fibrosis	IFN gamma	Subcutaneous	50 μg 3 X week	Reduction	Fever
	PTX + Vit E	Oral	400 mg 3 X daily + 300 mg 1 X daily	Reduction	

(1) Experience in the management of the manifest stage of CRS is limited to radiotherapy patients. In these conditions, an erythematous and erosive condition occasionally occurs, that is often associated with a burning itch. Treatment with loratadine, a non-sedating and mast-cell-stabilizing antihistamine, induced a marked relief of these symptoms and a shortening of the erythematous phase. Topical steroids generally have been used with success. Additional treatment modalities that have been reported to be of value in the manifest stage are cleansing of the oral cavity and administration of pilocarpine for prevention of mucositis. Heparinization and antibiotic prophylaxis for bacterial and viral infections may be beneficial.

(2) Treatment modalities for the chronic stage of the CRS were developed from Chernobyl sequelae and from therapeutic irradiation patients. Chernobyl patients responded well to a basic therapy with a linoleic acid ointment that blocked transepidermal water loss. Symptomatic telangiectasias disappeared after treatment by Argon laser. Tretinoin cream 0.005 percent applied once daily, led to clearance of focal and patchy radiation keratoses. Tretinoin cream appeared to cause more irritation than is common in patients with actinic keratoses. Intermittent anti-inflammatory treatment with topical nonatrophogenic steroids (mometasone buroate) was necessary. In more extensive lesions, oral application of the retinoid Acitretin (0.1-0.2 mg/kg daily) was used, analogous to the reported treatment of radiation-induced keratoacanthomas.

(3) Subcutaneous administration of interferon (IFN) has been beneficial to patients with severe and extensive radiation fibrosis (IFN gamma, 50 μg subcutaneously three times per week for 18 months). Using a protocol for scleroderma patients, fibrosis may be reduced almost to the level of uninvolved contralateral skin. Side effects included low grade fever to 38.5°C after the first two injections. The efficacy of IFN gamma may be explained in part by its antagonistic effect towards the cytokine TGF-beta, which is of importance for the induction of radiation fibrosis. Another therapeutic option for radiation fibrosis is the combined administration of pentoxifylline (PTX) (400 mg three times daily and Vitamin E 400 mg once daily). This regimen, applied for a minimum of 6 months, ameliorated persistent radiation fibrosis that had been progressive for over 20 years. Topical dressings of tetrachlorodekaoxide (TCDO) induce considerable granulation and re-epithelization in erosive skin conditions. Radioprotective properties of TCDO have been reported in experimental models that also demonstrated regenerative capacities in complicated wounds.

(4) Appropriate surgical procedures include excision of ulcers and contractures, wound closure by split and full thickness skin grafts, and in certain instances, vascularized flaps. Grafts usually heal without complications, including situations where the surrounding tissue may be affected by late radiation effects. The surgical experience, derived from patients with skin fibrosis after deeply penetrating radiation therapy, was that skin grafts do not heal if the surrounding affected tissue is not completely removed. This proved to be inappropriate for the survivors of the Chernobyl accident.

6-46. Chronic Radiation Syndrome

Chronic radiation syndrome (ChRS) has only been described as a distinct clinical entity in Russia and other former Soviet Republics. For example, when the Soviet Union developed its nuclear weapons program during and after World War II, safety procedures were often short-circuited in an effort to accelerate the production of plutonium. Workers in the first plant, the Mayak Plutonium Production Association, were often exposed to radiation at annual doses of 2.0 to 4.5 Gy. The diagnosis of ChRS was made in 1,596 workers. This entity was defined as a complex clinical syndrome occurring as a result of the long-term exposure of the organism to single or total doses of radiation which regularly exceed the dose permissible for professional exposure. The clinical course was marked by neuroregulatory disorders, moderate to marked leukopenia

(both neutrophils and lymphocytes depressed), and thrombocytopenia. In severe cases, anemia, atrophic changes in the GI mucous membranes, encephalomyelitis and infectious complications due to immune depression were noted. It is generally considered that persons who have been exposed to radiation for at least 3 years and have received at least 1 Gy or more to the marrow, and whose above signs and symptoms cannot be explained by other concurrent disease, may be considered to have ChRS.

a. Chronic suppression of the immune system by a prior radiation exposure has been documented. These deterministic, somatic responses are, by definition, predictable in nature, and reflect a change in blood-forming capacity following chronic radiation exposure. These changes have been noted both clinically in fractionated radiotherapy (ankylosing spondylitis patients), as well as epidemiologically in the recent reports of the health effects of Techa River area inhabitants. With chronic exposures in the Techa River area, a sizable incidence (about 3 percent) of ChRS developed. Chronic radiation syndrome is characterized by a hematologic syndrome, defined in terms of a chronic suppression of bone marrow function as manifested by a chronic leukopenia and thrombocytopenia. This, in turn, appeared to predispose affected individuals to increased risk to a variety of illnesses, including infections, degenerative diseases, and neoplasias.

b. Clinical symptoms are diffuse and may include sleep and/or appetite disturbances, generalized weakness and easy fatigability, increased excitability, loss of concentration, impaired memory, mood changes, vertigo, ataxia, paresthesias, headaches, epistaxis, chills, syncopal episodes, bone pain, and hot flashes. Clinical findings may include localized bone or muscle tenderness, mild hypotension, tachycardia, intention tremor, ataxia, asthenia, hyperreflexia (occasionally hyporeflexia), delayed menarche and underdeveloped secondary sexual characteristics. Laboratory findings include mild to marked pancytopenia and bone dysplasia. Gastric hypoacidity and dystrophic changes may be present. Once the patient is removed from the radiation environment, clinical symptoms and findings slowly resolve, and complete recovery has occurred from the lower doses.

c. Chronic radiation syndrome is not a condition that is likely to affect military personnel in operational settings since prolonged deployments to heavily contaminated areas, or long-term ingestion of highly contaminated food or water would be unlikely. At present, only a few places on earth would afford this opportunity (the area immediately surrounding Chernobyl and small areas at nuclear weapons test sites in Nevada; the Pacific Ocean; Semipalatinsk, Kazakstan; and Novaya Zemlya, Russia). A surface or near-surface nuclear weapon burst, a radiation dispersion device, a major reactor accident, or a similar event that creates contamination with high dose rates, given prolonged exposure, would permit development of this syndrome. Medical officers should be aware of this putative syndrome in order to properly counsel military personnel.

d. There may be sizable risks to long-term health problems of personnel involved with military operations within a radiologically contaminated environment. Those long-term health problems encompass not only cancer, but also other non-neoplastic diseases that contribute to increased morbidity such as chronic immune suppression and long-term injury to pulmonary, skin, skeletal, and reproductive tissues. The frequency and severity of these late

developing disease entities are exposure-dependent, regardless of their stochastic or deterministic nature. Such exposure needs to be considered in light of dose and dose-distribution within the targeted tissue and organ systems at risk. Application of molecular tools will allow radiation-associated injuries to be assessed and tracked within the individual, thus allowing for early detection and treatments to be employed.

Section IX. PSYCHOLOGICAL EFFECTS OF NUCLEAR WARFARE

6-47. General

Psychological stress can mimic the early symptoms and signs of acute radiation injury. Gastrointestinal symptoms (nausea, vomiting and diarrhea), fatigue, and headaches were frequently seen symptoms during episodes of *battle fatigue* in World War II. Long-term effects can also be expected, similar to the epidemics of chronic medically unexplained physical symptoms (MUPS) seen following Vietnam (Agent Orange Syndrome) and the Gulf War (Gulf War Illness).

6-48. Radiation Dispersal Devices

Radiation dispersal devices are devices designed to spread radioactive material over a wide area using conventional explosives rather than a nuclear detonation, or using other means to distribute radioactive material. The material released can pose a chemical hazard as well as a radiation hazard to personnel within a given contamination region. Although RDDs lack the destructive power of a nuclear detonation, the psychological impact of these devices might impede military operations by denying key terrain or installations and by degrading unit morale and cohesiveness. If an RDD attack occurs in a civilian setting, the psychological factors will be multiplied.

a. Psychological Effects of Radiation Dispersal Devices

(1) Fear of radiation and misunderstanding of its dangers is evidenced by the difficulties in the acceptance of nuclear industries. The use of an RDD would be expected to produce acute anxiety effects, including psychosomatic effects such as nausea and vomiting. Symptoms of acute radiation sickness in just a few personnel might trigger an outbreak of similar symptoms in the unit and/or in the civilian populace. The lack of information about the physical hazards of radiation increases the likelihood of military and civilian personnel responding with acute fear and anxiety from their perception of the threat regardless of the actual physical hazard posed by the RDD. An additional problem would be the strong possibility of an epidemic of chronic MUPS. In the face of MUPS, individuals who believe they were exposed tend not to believe governmental pronouncements of safety. The political problems that may ensue can overshadow the true medical impact.

(2) The severity of the psychological effects of an RDD will depend on the nature of the RDD material itself and the method of employment. A point source RDD that is left in an area of traffic but not dispersed by an explosive produces physical injury only to personnel within its immediate vicinity. There have been reports of significant injury where radiation sources were buried in abandoned military sites. Industrial accidents that have occurred may also be instructive. An accident involving an industrial radiography source led to radiation injuries in some of the exposed personnel as well as psychological effects in both exposed and unexposed personnel. In these instances, both real and imagined illnesses were attributed to radiation exposure. An RDD that uses a conventional explosion as a dispersal method will cause blast injuries in addition to radiation effects. There would also be the possibility of psychological casualties in personnel involved in the rescue work. Both types of RDD can also have psychological effects that can affect not only those injured, but also those that are uninjured physically. The greater number of casualties from the blast and a generally more frantic situation will intensify the level of stress on military personnel. Chemical toxicity from inhalation and wound contamination might lead to greater psychological effects.

b. Incidence.

(1) Normally, battle fatigue casualties are proportional to the numbers of wounded in action, which in turn are indicators of battle intensity. Radiation can be expected to increase the number of battle fatigue casualties. A lack of information on both radiation exposure and effects of exposure to an RDD contribute to battle fatigue. The amount of training as well as the intensity, duration, and degree of involvement in an RDD scenario will also contribute to combat stress. The number of battle fatigue casualties will also depend on the leadership, cohesiveness, and morale of a unit. Positive combat stress behaviors, such as altruism, heroism, and loyalty to comrades, may occur more frequently in units with exceptional *esprit de corps*.

(2) The long-term effects of possible chemical exposure may cause military personnel to suffer from long-term psychological stress (for example, Agent Orange Syndrome). A similar chronic psychological stress pattern could be expected to arise from the uncertainty about the effects of exposure to an RDD. Some of the potential effects include phobias, depression, and post traumatic stress disorder. Varieties of MUPS may arise as a result of uncertainty and anxiety about radiation effects, and the presence of MUPS may cause difficulties in diagnosis, especially in already complex cases of combined injuries.

(3) The use of an RDD within a civilian population center may produce more detrimental psychological effects to military personnel than use against a strictly military target. Currently, the military has seen increased peacetime operations, where closer relationships may exist between civilians and military personnel. Requests for treatment of civilian casualties, especially women and children, after exposure to an RDD might markedly increase the psychological impact on military personnel. Outbreaks of MUPS in civilian populations may result from fear of the effects of radioactive material. The mass casualty situation could severely overload emergency military operations and increase distress in military personnel.

6-49. Nuclear Detonation

A nuclear detonation presents intense stimuli, in that personnel witnessing the event are likely to suffer sensory overload as well as the fear of injury or death. Depending upon the yield of the weapon and the distance, the observer may see a brilliant flash that temporarily blinds him, hear a deafening explosion at incredible decibels, suffer thermal injury, feel the shock of blast winds, and then experience the ground quaking beneath his feet. At night, flash blindness could affect personnel miles beyond the range of any other acute effects. Such personnel may be highly stressed by these overwhelming stimuli, even in the absence of actual physical injury. The recognition that these signs indicate a nuclear blast may precipitate immediate adverse psychological reactions.

a. Contrary to media portrayals of disasters, mass panic is rare in disaster situations. It seems to occur primarily in situations where there are limited avenues of escape and possible entrapment, such as mine fires, sinking ships, or fires in crowded areas where exits become blocked by sheer numbers. The most frequent psychological effect after disasters is a temporary emotional disruption where people are stunned or dazed. This transient response may last minutes to days. Typically, such individuals will be able to respond to strong leadership and direction. Another psychological response is to become more efficient in the face of danger. This response is more likely in well-trained units with high morale. A third type of response would be that of a psychological casualty, where the transient emotional disruption is continued and more severe. Reactions include stunned, mute behavior, tearful helplessness, apathy, inappropriate activity, and preoccupation with somatic symptoms (often of emotional origin).

b. Somatic effects such as nausea, vomiting, diarrhea, and a feeling of weakness or fatigue would be likely to occur, especially in the psychological casualties. These individuals may exhibit helpless, aimless, or disorganized behaviors. In the aftermath of the only nuclear bombings to occur in wartime, some people were stunned into meaningless, repetitive behaviors with no obvious goal orientation or survival value. Some wandered uselessly in the debris, with no conscious effort to either escape or aid others. Many withdrew into an apathy approaching catatonia, apparently shutting themselves off from the outside world they felt unable to deal with or even face. They became psychically numb, unresponsive, and exhibited psychological dissociation evidenced by the so called *thousand yard stare*. In a region of widespread nuclear devastation and death, survivors may also suffer intense feelings of guilt, which would add more stress. Such guilt feelings are not based on an objective rational assessment of one's role in causing the deaths.

6-50. Fallout Field

Radioactive material may be deposited on ground surfaces after the use of nuclear weapons, RDDs, destruction of nuclear reactors, nuclear accidents, or improper nuclear waste disposal. Traditionally, the fall-out field contains the residual radiation that follows a nuclear detonation and consists of fission products, unspent fuel, and induced radioactive materials. The popular definition has been extended to include other sources of radioactive contamination, such as those resulting from a RDD, or the release of radionuclides from a nuclear power plant (either

accidental or intentional). The radioactive material in the area may be a significant hazard to personnel and could severely disrupt or halt military or civilian operations within this region. The true physical threat to personnel is proportional to the level of radioactivity and the length of time spent in the area. The locations of significant hazards or *hot spots* and the boundaries of a contamination field can change according to weather conditions and the type of terrain. Difficulty in detecting these hazards and the changing parameters of the field may increase the psychological impact on personnel.

a. The most stressful effects of a contamination field might be the uncertainties of both the radiation exposure and the perceived acute and chronic effects of radiation. The difficulties in providing accurate definition of the boundaries of a contamination field are an additional source of psychological stress. A chronic level of high stress will exist when monitoring an area for radiation hazards due to the perceived threat. Stress in this situation resembles that of engineers clearing an area of mines or patrolling a booby-trapped area. Presence of the radiation hazard may not be known until a unit enters the contaminated area. Military personnel may not know their individual exposure as only unit dosimetry may be available. This could become an issue when military personnel need to cross a contaminated field. The personnel may fear that they are getting a much larger exposure than deemed wise, especially if there is a lack of trust in the leadership. Re-suspension of hazardous material into the atmosphere from vehicles or military personnel stirring up dust in a contaminated area can increase the inhalation hazard. The feeling of being in danger but not being able to strike back, along with a lack of information, may contribute to stress levels and the possible development of battle fatigue symptoms.

b. Stress levels can be decreased with positive identification that defines the contamination field and with proper training of military personnel as to the hazards and effects. The specific psychological impact on troops will depend on the type of radiological hazard as well as the magnitude and dynamics of the boundaries of the contamination field. For example, plutonium has been frequently referred to as *the most toxic chemical on earth* by the popular media, and thus might be more likely to cause fear and anxiety in troops than other radionuclides. The degree of uncertainty regarding the exact location of hazards at any given time will be expected to produce anxiety and fear. The use of protective gear (mission-oriented protective posture [MOPP]) has shown to produce stress in military personnel and degrade the performance and communication capability of a unit, if the unit is not properly trained.

c. The most extreme psychological damage occurs when physiological symptoms from an unknown toxic exposure become manifest. Significant degradation in performance may occur as military personnel become concerned about to what they were exposed, the dose, and the future effects of that exposure. An increase in the prevalence of battle fatigue could occur in this situation. Proper detection of a contamination field and education of troops before the onset of symptoms is, therefore, the most effective method to ensure successful accomplishment of a unit's mission.

6-51. Psychosocial Sequelae of Radiation Exposure

a. Psychosocial Sequelae. Even in the absence of actual exposure, fear that one has been exposed to radiation may cause psychosocial sequelae. Since fear and anxiety are stressors, the person may experience psychosomatic symptoms, some of which may mimic early acute radiation syndrome features. For example, in the incident at Three Mile Island in 1979, surveys of the surrounding population found an increase in such psychosomatic symptoms as nausea, anorexia, and skin rashes, even though there was no detectable radiation release in most of that region. At Goiânia, Brazil, after scavengers opened a medical radiotherapy device containing radiocesium, approximately 5,000 of the first 60,000 persons (8 percent) to be screened for radioactive contamination showed symptoms of acute stress or allergies such as a rash around the neck and upper body, vomiting, and diarrhea. However, none of these individuals were contaminated. Thus, the perceptions and preconceptions about a radiation accident may be just as important as the radiation itself in terms of subsequent pathology.

b. Psychological Factors at Chernobyl.

(1) Many of the recovery team members, *liquidators*, called in to help with the cleanup of the reactor at Chernobyl were military personnel. A study of Estonian liquidators found no increases in cancer, leukemia, or overall mortality, but they did find an increase in suicide. A study of Latvian liquidators found that almost half had psychosomatic disorders. Part of the uncertainty for the liquidators was a distrust of the Red Army's record of the radiation doses. The fear of radiation in the liquidators was probably enhanced both by their lack of knowledge and the misinformation in the media. An epidemic of *vegetative dystonia* occurred in liquidators and people from the contaminated areas (see Appendix D). The vegetative dystonia was more prevalent in liquidators who suffered acute radiation sickness, but is also seen in others who suffered no acute effects. The symptoms of vegetative dystonia resemble the MUPS seen in Agent Orange Syndrome and Gulf War Illness, as well as neurocirculatory asthenia or *effort syndrome* that was prevalent during and after both World Wars.

(2) Many people living upwind of Chernobyl and hundreds of miles away received detectable doses of radiation equivalent to or less than a doubling of the normal background radiation level. Some people became so afraid of the fallout that their whole lives began to revolve around avoidance. Whenever possible, they refused to go outside or eat locally grown produce. Some sank into deep despair and committed suicide rather than risk what they believed would be the inevitable and horrible effects of radiation. Such severe reactions were referred to as *radiophobia* by the media.

(3) Psychiatric disorders in Belarus were estimated to be present in 35.8 percent of the population 6 years after Chernobyl. Most prevalent were affective disorders (16.5 percent) and anxiety disorders (12.6 percent). These were not evenly distributed within the population, but were more common among evacuees and mothers with young children. Other studies found that people living with partners were better able to cope than those living alone. Thus, the more people were personally threatened or affected by the incident, the more likely they were to develop psychosocial sequelae. However, those with social support were better able to handle the increased stress and resist such consequences.

6-52. Treatment.

a. The treatment of psychological stress resulting from actual or perceived exposure to radiation is the same as that for battle fatigue. The acronym PIES (proximity, immediacy, expectancy, simplicity) can be used to remember the treatment principles. Proximity means to treat the battle fatigue casualty as close as possible to the unit and the battle from which he came, so as to minimize overevacuating a casualty. Immediacy refers to initiating treatment as soon as possible, to prevent the strengthening of maladaptive habits and the self-perception of illness or disability. Expectancy means that all staff should convey the positive expectation that the casualty will fully recover and be able to return to duty after a short respite from the battle. Simplicity refers to the use of simple, brief, and straightforward methods to restore physical well being and self-confidence. In addition to PIES, military personnel should also be given more information about radiation and radiation effects. This should improve their outlook and help stop the spread of wild rumors.

b. Sedatives or tranquilizers should be avoided unless they are essential to manage sleep or agitated behavior. Stress casualties should only be evacuated to the next higher echelon if their symptoms make them too disruptive to manage at a given echelon. Similarly, hospitalization should be avoided unless absolutely necessary for safety. Those requiring brief hospitalization should be transferred to a nonhospital treatment setting as soon as their condition permits.

6-53. Prevention and Risk Communication

a. Prevention. Prevention, when possible, is always superior to treatment. Prior to an engagement where the use of nuclear weapons or RDDs is possible, the medical staff can suggest and implement programs for line commanders to instruct their units about radiation and its effects. In general, troops who are psychologically prepared for specific stresses are better able to endure them and will suffer fewer and less severe adverse reactions. This same principle is widely used in preparing troops to cope with MOPP gear, chemical agent exposure, and other adverse environments. Postexposure training will be much less effective.

b. Risk Communication.

(1) Effective risk communication depends upon a number of factors including trust in unit leadership. Furthermore, the training itself should be--

- Tailored to the cognitive level of personnel so that they can understand it.

- Realistic and accurate.

- Practical rather than theoretical, with concrete suggestions on self-protection. In the case of potential radiation exposure, practical suggestions should emphasize the use of time, distance, and shielding to minimize exposure.

(2) Specifically included in all training programs should be sections on normal levels of radiation exposure including the levels incurred by patients undergoing medical evaluation. Mission exposure levels should be explained in the mission planning. Cancer risks should be presented directly to those involved in view of national cancer occurrence risks and level of increased risk due to specific levels of exposure.

(3) Leadership should recognize the importance of keeping troops informed of possible exposures and the importance of reassuring the personnel that medical treatments exist to reduce the effects of actual exposures. The soldier who believes any amount of exposure to radiation, no matter how slight, can be life-threatening is far more likely to have adverse effects than the one who understands that low levels can be harmless and that even intermediate levels can be successfully treated.

Section X. LOW-LEVEL RADIATION

6-54. General

a. As mentioned in Chapters 1 and 2, current military operational exposure risks involve primarily terrorist actions with improvised nuclear devices, RDDs, nuclear accidents, and contamination rather than the risks associated with tactical or strategic nuclear war. Therefore, the risk of exposure to low-level radiation is more limited geographically and the immediate exposure risks to soldiers might be much lower. Except in rare circumstances, the radiation doses received under these types of operations would be well below those that would cause serious radiation injuries soon after exposure. However, they would be above the occupational dose limits that are applied to civilian workers and military personnel assigned to routine nuclear operational duties.

b. Emphasis has also shifted from helping military personnel to survive immediately disabling and life-threatening injuries to medical care and follow-up for delayed health effects of low-level radiation, such as the development of radiation induced cancer. This section will briefly cover proposed low-level radiation exposure guidance, documentation of medical records, medical care for exposed personnel, and long-term medical follow-up.

6-55. Exposure Guidance

a. In peacetime while not deployed, radiation exposures of soldiers whose normal operational duties include, for example, handling of military radioactive commodities, are governed by DOD radiation protection regulations. These regulations are comparable to civilian worker protection regulations. However, this exposure guidance is generally not applicable for nonoccupational, low-level exposures, such as a maneuver unit moving into a potentially hazardous area during stability operations and support operations.

b. The occupational annual dose limit is 50 mSv, while 700 mSv is the threshold for the development of acute health affects that become a concern in nuclear war. The risks associated with radiation exposure within this range of 50 to 700 mSv are confined primarily to the risk of increased incidence of malignant diseases, including solid tumors and leukemias. The lack of exposure guidance between these two limits has just recently been addressed by Army and NATO planners, and the guidance is presented here for consideration by other services for guidance on low level radiation safety. The US Army Nuclear and Chemical Agency coordinated a joint service meeting and established recommended revised exposure guidance. The revised Operational Exposure Guidelines are shown in Table 6-21.

Table 6-21. Revised Low-Level Radiation Guidance for Military Operations

Total Cumulative Dose [a]	**Radiation Exposure State Category**	**State**	**Recommended Actions**	**Increased Risk of Long-Term Fatal Cancer** [b]
0-0.05 cGy	0	NO RISK	- None	None
0.05-0.5 cGy	1A	NORMAL RISK	- Record individual dose readings. - Initiate periodic monitoring.	1:4,000
0.5-5 cGy	1B	MINIMAL RISK	- Record individual dose readings. - Continue monitoring. - Initiate rad survey. - Prioritize tasks. - Establish dose control measures as part of operations.	1:400
5-10 cGy	1C	LIMITED RISK	- Record individual dose readings. - Continue monitoring. - Update survey. - Continue dose control measures. - Execute priority tasks only. [c]	1:200
10-25 cGy	1D	INCREASED RISK	- Record individual dose readings. - Continue monitoring. - Update survey. - Continue dose control measures. - Execute critical tasks only. [d]	1:80
25-70 cGy	1E	SIGNIFICANT RISK	- Record individual dose readings. - Continue	1:30

			monitoring. - Update survey. - Continue dose control measures. - Execute critical tasks only. [d]	

[a] The use of the measurement millisievert (mSv) is preferred, however, due to the fact that normally the military has only the capability to measure centigray (cGy), US Forces will use cGy (as long as the ability to measure in mSv is not possible). For whole body gamma radiation, 1 cGy is equal to 10 mSv. All doses should be kept as low as reasonably achievable. This will reduce the risk to individual service members and will retain maximum flexibility for future operations.

[b] This is in addition to the 1:5 and 1:4 incidence of fatal cancer among the general population. Increased risk is given for induction of fatal cancer (losing an average of 24 years of life for personnel ages 20 to 30 years). Total lifetime risk is assumed to 4 to 7 percent per 100 cGy (1000 mSv). It must be noted that higher radiation dose rates produce proportionally more health risks than the same total dose given over a longer period.

[c] Examples of priority tasks are those missions to avert danger to persons or to prevent damage from spreading.

[d] Examples of critical tasks are those missions required to save lives.

6-56. Documentation of Radiation Exposure Records

The operational exposure guidance (OEG) concept requires that all units maintain radiation exposure records. In the US Army, the records are based on platoon-level data received daily, or after a mission in a radiological contaminated area. The unit dose is an average of the doses to individuals in the unit who have dosimeters, usually two per squad in the US Army. Therefore, the US Army assumes that each soldier receives an individual dose equal to that of the average for the platoon. When a soldier transfers out of an exposed unit, the RES for that platoon is noted in the soldier's personnel file. When possible, soldiers are reassigned to platoons with the same RES category. Although this might create personnel strength management problems, it is intended to prevent personnel from incapacitation due to overexposure to radiation in future operations.

6-57. Medical Care

Medical care following exposure to low-level radiation involves the management of both the early and delayed deterministic events from doses above threshold levels (bone marrow depression, skin injuries), and the management of stochastic effects, primarily nonspecific tumors that may become clinically evident years after exposure to radiation. Within the low-level dose range (50 to 700 mSv), the greatest risk is the appearance of stochastic effects, that is, the appearance of benign and malignant tumors years after the event. However, because of the uncertainty of the dose that may be received during certain situations, deterministic effects that will appear within months of certain types of acute exposures will be briefly discussed.

Examples of scenarios in which US personnel may become involved with a risk of exposure to low-level radiation include--

- Responding to a nuclear reactor incident.
- Securing a negligently or deliberately abandoned sealed radiation source.
- Participating in containing radioactive materials exposed to the environment, as may occur if a nuclear waste dump is disturbed.

These types of events could occur during the performance of routine home station duties, during missions in hostile or nonhostile territory, or while responding to terrorist actions. Exposures may be acute or chronic, they may involve nonuniform irradiation resulting in high doses to specific areas of the body, they may occur alone or with radioactive contamination, and they may occur with or without trauma or other injuries or illnesses.

a. Early and Delayed Deterministic Effects.

(1) It is unlikely that symptoms of deterministic effects will appear due to acute whole or partial body radiation doses of less than 1 Sv (100 rem). Early evidence of acute radiation-induced cellular injury, for example, structural changes in the chromosomes of some circulating lymphocytes, and falls in the absolute lymphocyte and sperm counts, is, however, clinically detectable in asymptomatic individuals who have received lower doses.

(2) Expression of acute radiation injury in some cell systems is delayed for weeks or months after an acute exposure to penetrating radiation above threshold levels. A period of transient male infertility may follow exposure to doses at the upper end of the 50 to 700 mSv range. After higher but sublethal whole body doses, males will experience a low sperm count with a nadir at about 45 days postexposure or an absence of sperm for a postexposure period that is directly proportional to the dose. Females may experience a period of amenorrhea after acute radiation exposure. Several Sv to the gonads are required to cause permanent sterility in previously fertile males and females of reproductive age; thus, sterility will not be a problem for individuals at risk of doses in the 50 to 700 mSv range. The threshold doses for the typically delayed (for weeks or months) expression of acute radiation injury to other tissues--such as the endothelial cells lining the blood vessels and connective tissue and their replacement by fibrous tissue (fibroatrophy), the optic lens (cataract), and the thyroid gland (thyroid hypofunction) - also are considerably higher in the range of the 50 to 700 mSv. Therefore, specific medical care for these conditions would fall under procedures outlined in earlier sections of this chapter for higher doses of radiation exposure.

b. Stochastic Effects. The primary stochastic or late effect of exposure to radiation is the development of radiation-induced tumors of types that are not caused only by exposure to radiation; they may be benign or malignant. It is assumed that such tumors will become clinically evident among the population some years after exposure to radiation. The probability of this occurring is directly related to the dose received. Radiation-induced or radiogenic tumors are histologically and clinically indistinguishable from spontaneously occurring tumors. Their

diagnosis, treatment, and management are the same as those for spontaneously occurring cancers of the same type.

6-58. Medical Follow-Up

The low-level dose range (50 to 700 mSv) is unlikely to cause delayed acute or chronic deterministic effects. Therefore, this paragraph will address medical follow-up actions involved with the main long-term effect of radiation exposure--malignant disease. Past extensive research has identified certain malignant diseases that can be induced by radiation as well as by other agents. Those malignant diseases that have been associated with prior radiation exposure are termed radiogenic. These include the following:

- All types of leukemia except chronic lymphocytic leukemia.
- Female breast cancer.
- Cancers of the lung, stomach, thyroid, esophagus, small intestine, colon, liver, skeleton, CNS, salivary glands, and ovary.
- Nonmelanoma skin cancer.
- Non-Hodgkin's lymphoma.
- Multiple myeloma.

a. Medical Assessment. Medical assessment is the evaluation of basic parameters of general and radiological health status after a known or suspected exposure to radiation or radioactive contaminants. Such an evaluation may be prompted by the development of nonspecific symptoms, trauma, or an observed degradation of individual performance during or after a military operation conducted in an area of known or suspected radiation or radioactive contaminants. Personnel are not likely to develop symptoms of acute radiation exposure at the low-level dose range; however, medical assessment is recommended after personnel exit areas of potential exposure. The purpose of the assessment of asymptomatic individuals in these situations is to--

- Rule out that personnel were exposed to higher than expected doses.
- Obtain baseline clinical data to assist in estimating the individual's radiation dose.
- Establish a basis for recommendations regarding the individual's need for medical care, periodic monitoring, or specific testing.

b. Medical Monitoring.

(1) Medical monitoring is a systematic screening of a population of asymptomatic individuals for preclinical disease with the purpose of preventing or delaying the development and progression of chronic disease in those individuals. However, medical monitoring after radiation exposure is not routinely suggested or practiced for individuals with known or suspected exposures to radiation. An exposure or a presumed exposure to radiation is not, by itself, sufficient to justify a medical monitoring program. The decision about whether a medical monitoring program is appropriate and necessary in a given situation should be based on consideration of a number of factors including a rigorous cost-benefit analysis. This analysis should take into account the following considerations:

- The certainty, type, intensity, and duration of the dose concerned.
- The history and population prevalence of the disease concerned.
- The effectiveness, sensitivity, specificity, and potential hazardous side effects of available screening tests.
- If test results are positive, the availability, benefits, and risks of treatment protocols.

(2) The latent period between radiation exposure and the development of a clinically detectable tumor may have an effect on the design of a screening program. For the US Armed Forces, personnel are usually between 20 and 40 years of age when they are exposed, and most radiation-induced tumors would be expected to become clinically evident when they are older than 40, and in most cases, older than 50. Since most cancers occur spontaneously at older ages (older than 50 years) without exposure to radiation, few tests have shown to be of benefit in terms of improving either survivability or quality of life. Tests that have been recommended include the Pap smear and mammography. Since the risk of cancer in nonexposed populations is high over a normal lifetime, the risk of radiation-induced tumors due to exposure to low-level radiation would almost always be far less than the risk of normal spontaneous incidence. Therefore, it is the spontaneous cancer risk, not the radiogenic cancer risk that should drive the decision to conduct a monitoring program.

6-59. Health Effects of Exposure to Depleted Uranium

While many Gulf War veterans have reported an array of physical and mental health complaints since the war, it is not yet clear the extent to which veterans are experiencing either higher than expected rates of identifiable illnesses with known etiologies or any other illnesses from as yet unidentified origins. Since the Gulf War was the first time there was widespread use of DU, there is very little direct experience with the health effects of DU. However, there are extensive experiences and a wide body of literature dealing with the health effects of natural and enriched uranium. The toxicological effects of natural uranium are identical to those of DU, while the radiological effects of DU are always less pronounced because DU is less radioactive than natural uranium. In general, heavy-metal toxicity is regarded as posing a more serious health risk than its radiation. This is because DU produces a low level of radiation per unit mass. The most

abundant isotope in natural uranium, ^{238}U, has a very long half-life (4.5 billion years), which means that it decays slowly and thus produces fewer disintegrations per mass than an isotope that decays rapidly, such as ^{234}U (half-life of 245,000 years). As mentioned, DU is less radioactive than natural uranium and, indeed, is classified in the lowest hazard class of all radioactive materials. (See Chapter 2 for a detailed discussion of the properties of DU.)

a. Internal Exposure. In nonmilitary situations, the main routes of uranium uptake by the human body are inhalation and ingestion, as is the case with other heavy metals. In the military environment, additional routes of uranium exposure exist such as from embedded metal fragments slowly dissolving in the body and uranium-contaminated open wounds. All of these routes of internalization contribute to the total body burden of uranium. In addition to being very dense (almost twice as dense as lead), DU, like any uranium, is pyrophoric, that is, in fine particles it can ignite easily. When a DU penetrator strikes armor or burns, it produces uranium dusts or aerosol particles, which can be inhaled. From 10 to 35 percent of the original material is aerosolized and approximately 60 to 69 percent of the aerosolized fraction is respirable. From the heat of combustion as well as weathering, these small particles will eventually become oxidized, forming predominately depleted U_3O_8 but also small amounts of depleted UO_2 and depleted UO_3. Most of the suspended aerosols will rapidly settle to the ground. Activity or surface winds may disturb the settled particles and resuspend and redistribute a fraction of them. Once internalized, a fraction of the particles dissolve and enter the bloodstream, where most uranium is excreted from the body through the kidneys.

(1) The schematic in Figure 6-6 depicts how uranium interacts with the body. Inhaled, ingested, or embedded fragments reach the blood after solubilizing either at the site of entry or at some other location in the body where they end up. For instance, some inhaled uranium enters the blood from the lungs, and some of the uranium originally in the lungs ends up in the GI tract as a result of mucociliary clearance from the respiratory tract and subsequent swallowing. Uranium then accumulates to some degree in all organs. The major portion of uranium in blood is excreted in the urine, with the remainder distributed mostly to bone and soft tissue. There are few data to show the content and distribution of uranium in human tissue from inhalation and ingestion from natural sources.

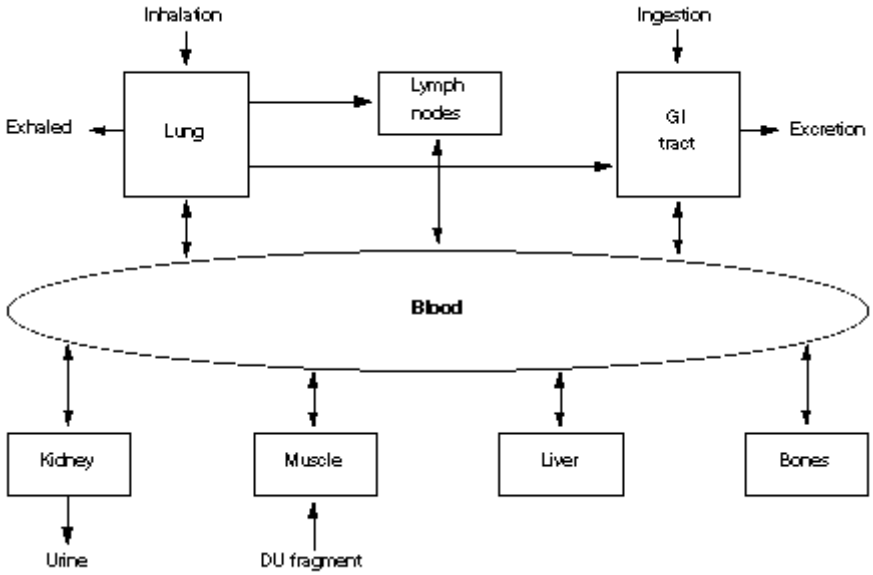

Figure 6-6, Depleted uranium internalization schematic.

(2) Following exposure to uranium and its distribution among various body compartments, or pools, uranium disappears from various tissues at different rates. The half-life in bone is approximately 300 days while in blood it is less than one-half day. In the lungs, retention of uranium depends on the solubility of the various oxides to which the lung is initially exposed. Even within some tissues, the uranium is distributed between compartments and exhibits a range of half-lives. Renal uranium, for instance, is lost at rates reflecting its presence in at least two distinct kinetic pools, one with a relatively short half-life of around 6 days and a second one with an indeterminate half-life. This conclusion is complicated by the likelihood that, as with other metals, the onset of renal damage will decrease the retention time of uranium.

b. Chemical Toxicity. Extensive information is available on the occupational exposure of workers in the uranium industry. These include workers in both uranium mines and in contractor facilities, where uranium was separated from the ore and uranium metal was produced for the enrichment process for nuclear weapons and nuclear fuel. Workers' exposure to uranium during the peak operational era was high in some cases. There were occupational guidelines and standards at the time; however, in many cases these standards were exceeded. Various studies report exposures of up to 9,000 to 10,000 µg natural U/m^3 at the Y-12 plant in Oak Ridge and at the Mallinckrodt plant (outside of St. Louis). No adverse health effects appeared in the uranium millers who worked with uranium and were exposed to levels of natural uranium far in excess of present standards.

(1) The kidney is considered the target organ for uranium for chemical toxicity because of DU ingestion. The primary renal site of action of uranium is the proximal tubule where proton secretion degrades the bicarbonate complex of the uranyl ion, permitting the uranium to react with apical cell membranes of the tubular epithelium. This view is supported by the observation that alkalinization of urine increases urinary uranium excretion. It has not been established whether the reaction between uranyl ions and the cell membrane directly

interferes with membrane function, as has been reported in yeast, or whether the primary target of the metal lies inside the renal cell.

(2) For inhaled uranium dust, there is, however, convincing evidence of excess lung cancer in underground uranium miners. It is well established that the carcinogenic agent in the mines was not the uranium ore but the gaseous decay products of radon-222. Radon has two alpha emitting short-lived decay products that form in the air and deposit efficiently on the bronchial surfaces. Two studies showed that the lung dose from inhaled uranium ore was too low to be of significance relative to the bronchial dose from radon decay products. The mines also had other airborne substances, such as ore dust, blast fumes, and diesel fumes (in later years only). Studies indicate that the relationship of lung cancer risk across a broad spectrum of other air contaminants was similar only to exposure to radon-222, and that no other substance could be implicated. Smoking enhanced the lung cancer risk in a submultiplicative manner such that for the same radon exposure, the risk of getting cancer among individuals who had ever smoked was twice as high as that of those who never had.

c. Radiological Toxicity. Negative health effects resulting from the radiation effects of depleted or natural uranium particles which have been inhaled have not been observed in humans. There is evidence of lung cancer in miners from epidemiological studies, but this is related to exposure to a combination of airborne short-lived decay products of radon and other air toxicants, such as silica dust, diesel fumes, and cigarette smoke (see paragraph 6-59b(2)). Uranium mill workers have not shown excess lung cancer or other diseases despite their increased exposure to uranium and radon progeny. A population of workers was exposed to insoluble uranium dust (including UO_2) at levels of 0.5 to 2.5 mg U/m^3 with some exposed up to an estimated 10 mg U/m^3 for about 5 years. None of these workers were exposed to other potential irritants. They did not exhibit respiratory disease.

(1) Lung damage resulting from inhalation of uranium oxides is usually noncancerous alveolar epithelium damage of type II cells. Animal studies have examined pulmonary damage from exposure to uranium oxides. Exposure to 5 mg UO_2/m^3 for more than 3 years did not result in damage to the lungs, but minimal fibrosis, suggestive of radiation injury, was occasionally observed in the tracheobronchial lymph nodes of dogs and monkeys and lungs of monkeys. Alpha radiation doses were estimated to have been greater than 500 rads in the lungs and greater than 7,000 rads in tracheobronchial lymph nodes. Lung fibrosis is consistent with relatively short-term high exposure rate in animal experiments.

(2) There is no evidence of cancer or any other negative health effect related to the radiation received from exposure to natural uranium, whether inhaled or ingested, even at very high doses. The biological properties of uranium in the body and its absorption from the GI tract are reasonably well known from occupational exposures, studies of normal environmental intake, and animal studies. The radiation dose can be calculated for any body organ given the amount in the organ. Based on the distribution in the body and the known body organ content, no health effects from radiation would be expected even for high occupational exposures. This results mainly because of the low radioactivity of natural uranium and the inability to get enough into the body to deliver a radiation dose that could be significant in causing cancer. The same would be true for DU.

d. Embedded Fragments, Wound Contamination, and Follow-up. Embedded DU fragments and wound contamination with DU dust is another pathway for exposure, seen almost exclusively in a combat environment. In combat use, DU metal fragments from a penetrator or a vehicle's hull armor can scatter inside the vehicle, killing and injuring personnel, destroying equipment, and causing secondary explosions and fires. As the use of DU munitions is relatively recent, there is little published literature on exposure to embedded fragments. In response to a recommendation from Armed Forces Radiobiology Research Institute (AFRRI), physicians and scientists from DOD and the Department of Veterans Affairs (VA) drafted a protocol to be used to follow-up soldiers affected by DU munitions. The protocol, reviewed and revised by a panel of experts, was submitted to the Army on 7 December 1992, and implemented in late 1993 as the DU Follow-Up Program at the Baltimore VA Medical Center.

(1) Currently, 33 participants are in the program. All were evaluated at the Baltimore VA Medical Center in 1993 and 1994, and 29 were reevaluated in 1997. Of those evaluated, about half have been identified radiographically as having retained metal fragments. In 1997, the majority of individuals identified radiographically as having retained DU fragments had elevated 24-hour urinary uranium levels. This suggests that DU is being oxidized in body fluids. Thus, these metal fragments are not entirely inert. Most individuals who did not have embedded fragments did not have elevated urinary uranium levels. Twenty-six DU exposed participants in the DU Follow-Up Program at the Baltimore VA, and 19 unexposed persons underwent whole body radiation counting (WBRC). A summary statistic based on the WBRC result was derived for each participant, and this correlated highly with the 24-hour urinary uranium result.

(2) Although these individuals have an array of health problems, many of which are related to their combat injuries, *to date no manifestations of kidney disease attributable to the chemical toxicity of DU have been found; neither do these individuals appear to have manifestations attributable to radiation effects.* However, several irregularities in biochemical and neuropsychological testing have been correlated with elevated urinary uranium, the clinical significance of which is unclear. These patients continue to be followed to be sure that this remains the case.

(3) Laboratory tests also found DU in semen in samples from some, but not all, veterans exposed to DU. This is not altogether surprising as DU disperses throughout the body. It is not clear what effect, if any, this might have on reproduction of a couple, where one partner has an embedded DU fragment. To date, all births to couples in the DU Follow-Up Program have been normal. There have also been no studies to determine the level, if any, of uranium from dietary intake appearing in semen in individuals living in areas with high levels of natural uranium.

(4) Participants in the DU Follow-Up Program at the Baltimore VA Medical Center also participated in a set of standard tests to measure neurocognitive function. As a group these individuals performed normally on standard tests of attention, memory, and problem solving. However, there was a statistical relationship between elevated urine uranium and lowered efficiency and accuracy measured in a follow-up computerized test. Researchers have

urged caution in looking at these results as *an analysis of individual cases suggested that the performance of a few patients might have been responsible for this relationship on this relatively small sample of patients with elevated uranium levels. There was no evidence of worsening performance over time.*

e. External Exposure. The primary external health hazard from DU is beta and gamma radiation generated by the radioactive decay of uranium decay products. The vast majority of DU radiation (more than 95 percent) is alpha radiation, which cannot penetrate paper or skin and has been shown not to cause adverse external health effects. Gamma and beta radiation from decay products can penetrate the skin, and in sufficient amounts, could pose a health risk. Intact munitions and armor have the potential for amassing sufficient DU to generate enough beta and gamma radiation to exceed occupational levels. However, all DU weapon systems are shielded to control gamma and beta radiation emitted from DU. As such, they present very little external exposure risk for personnel working with intact munitions and armor (see Chapter 2). Chemical toxicity does not pose a threat unless the metal is internalized.

f. Conclusions. From a review of scientific studies and literature, and from the recent experiences of Gulf war veterans, the following can be concluded:

- Although any increase in radiation to the human body can be calculated to be harmful from extrapolation from higher levels, there are no peer-reviewed published reports of detectable increases of cancer or other negative health effects from radiation exposure to inhaled or ingested natural uranium at levels far exceeding those likely in the Gulf. This is mainly because the body is very effective at eliminating ingested and inhaled natural uranium and because the low radioactivity per unit mass of natural and DU means that the mass of uranium needed for significant internal exposure is virtually impossible to obtain.

- External radiation takes the primary form of alpha radiation, but amounts of beta and gamma radiation also exist. Alpha radiation is not capable of penetrating cloth or skin and would therefore have no negative health effect. Beta and gamma radiation, which can have negative health effects, have been measured at levels below those expected to be of concern.

- Large variations in exposure to natural uranium in the normal environment have not been associated with negative health effects.

- Radiation-related effects from embedded fragments will depend on the size of the fragment and its proximity to vital organs.

- Exposure to uranium and other heavy metals in large doses can cause changes in renal function and at very high levels result in renal failure.

- In spite of these findings, no increased morbidity or frequency of end-stage renal disease has been observed in relatively large occupational populations chronically exposed to natural uranium at concentrations above normal ambient ones.

- The cohort of individuals, about half of whom have embedded fragments, who are being followed at the Baltimore VA Medical Center as part of the DU Follow-Up Program, represents a group of Gulf War veterans who received the highest levels of exposure to DU during the Gulf War. Although many of these veterans have health problems related to their injuries in the Gulf War, and those with embedded fragments have elevated urine uranium levels, researchers to date report neither adverse renal effects attributable to chemical toxicity of DU, nor any adverse health effects that relate to DU radiation. They do, however, note several biochemical perturbations in neuroendocrine parameters related to urinary uranium concentrations. In some subtle neuropsychological test findings; the clinical significance of this is unclear.

APPENDIX A

RADIATION DETECTION AND DOSIMETRY

A-1. General

The purpose of this appendix is to provide medical personnel with information on the basic principles of radiation dosimetry (physical and biological), and the types and principles of operation of radiation detection instrumentation. The detection and repeated measurement of radioactive areas produced by nuclear explosions, nuclear accidents, or RDDs will give important information affecting the operation of field medical units. In addition, medical personnel must be trained in the detection and measurement of patient contamination in order to prevent uncontrolled exposures of MTFs and personnel. Physical dosimeters provide a direct measure of the radiation energy deposited in the dosimeter and can be either self-reading or require processing for determination of dose received. The basic requirement of any radiation-measuring instrument is that the instrument's detector interacts with the radiation in such a manner that the magnitude of the instrument's response is proportional to the radiation effect or radiation property under measurement. The absorbed dose of ionizing radiation in exposure cases should be assessed in order to predict and treat the medical consequences. The absorbed dose and, depending on the level of irradiation, the fraction of the absorbed dose within the body, has to be determined with a sufficient degree of accuracy and precision. It is generally accepted that differences of ten percent in absorbed dose can produce clearly observable variations in biological response.

A-2. Physical Dosimetry

This paragraph discusses some of the operating principles and applications of various radiation detection and measurement instruments that may be used by the military. Definitions and descriptions of terms used in connection with radiation detection and measurement instruments are presented first. The operating principles and applications of selected instrumentation are then described.

a. Terminology.

(1) *Absorbed Dose.* The International System (SI) unit for radiation absorbed dose in any material is the Gy. The dose is the total amount of energy absorbed, and the exposure could be single or multiple and either short or long in duration. The term *rad* is the previously used unit for radiation absorbed dose. Since 1 Gy is equal to 100 rad, then 1 cGy is equal to 1 rad. Use of the term *Gy* is not restricted to X- or gamma radiation, but can be used for all forms of ionizing radiation including particulate radiation.

(2) *Dose Rate.* Dose rate is the dose of radiation *per unit time.* The old unit for this is the Ci, in honor of Marie Curie who discovered radioactivity while working with radium-226. The curie is based on the activity of 1 gram of radium-226, or 3.7×10^{10} radioactive

disintegrations per second. The SI unit for measuring the rate of nuclear transformations is the Bq.

(3) *Excitation.* Excitation is a change in the energy level of an orbital electron and occurs when the energy lost by the incident radiation is insufficient to cause ionization. It can result from interactions involving incident photons of gamma or X-rays or from inelastic collisions of particles. When the excited electron returns to its ground-state energy level, it must give off energy in the form of a photon of electromagnetic radiation, which is usually of low enough energy to be detected with a photomultiplier tube.

(4) *Ionization.* Ionization is the separation of an electrically neutral atom or molecule into electrically charged components termed an ion pair. This is accomplished by the removal of one or more electrons (negatively charged) from an atom or molecule, which then becomes a positively charged ion.

(5) *Specific ionization.* Specific ionization is the number of ion pairs per unit distance formed along the path of a particle, and is usually specified in centimeters (cm). The density of ionization produced by a given particle is a function of its charge and its velocity. A more slowly moving particle spends more time in the vicinity of the atom or molecule, increasing the chance of ionization occurring. As an example, because of its slowness and charge, an alpha particle will produce thousands more ion pairs per cm of travel than an electron (beta particle) of the same energy (approximately 100 ion pairs per cm).

(6) *Gas amplification.* The gas amplification factor is defined as the ratio of the number of electrons collected by the anode to the number of electrons formed by the primary radiation interaction. If sufficient potential difference is applied across a detector's electrodes, the free electrons resulting from ionization are accelerated toward the anode with enough energy to ionize the neutral gas atoms that are in their path, resulting in secondary ionization. This secondary ionization or amplification in the gas can add many thousands of free electrons to the sensitive volume for each primary electron that was formed by the radiation.

(7) *Pulse height.* Pulse height, which is a quantitative measurement of current flow, depends upon both the gas amplification factor and specific ionization of a given radiation. Since alpha particles have the highest specific ionization, they will produce the largest pulses. Beta particles, having a specific ionization of about 1/1,000 of alpha particles of equivalent energy, will produce pulses about 1/1000 the amplitude of those produced by alpha particles.

(8) *Free-in-air-dose.* The term *free-in-air-dose* refers to the radiation that would be measured in air at a certain point. This differs from other radiation doses of importance that might actually be desirable to measure, such as midline tissue dose or dose to the blood forming organs. The differentiation is made since free-in-air-dose is exceedingly easy to measure with current field instruments, while the more meaningful dose may be estimated or determined by use of a scaling factor or conversion approximation. Military tactical dosimeters measure free-in-air-doses.

b. Physical Dosimeters. Human senses do not detect ionizing radiation; therefore, special instrumentation must be used for radiation detection and measurement. The degree of hazard from radiation to humans depends on the type of radiation, its energy spectrum, as well as the quantity to which a person has been exposed. Therefore, radiation dosimeters used in the field must be capable of making qualitative as well as quantitative measurements.

(1) An ideal instrument for practical use in the field would have the characteristics listed below. No single instrument at present has all the characteristics described. Accordingly, different types of instruments must be used depending upon the nature of the radiation hazard.

- Measure dose, or dose rate, in units directly applicable to the tissue of concern.
- Respond to only one kind of radiation at a time.
- Have the desired sensitivity and accuracy, independent of the energy of the radiation.
- Be free of calibration and zero drift within reasonable times.
- Be free of extraneous effects from gravitational forces, temperature, humidity, chemical vapors, electrical and magnetic fields, shock, and so on.
- Have a short response or readout time.
- Indicate the occurrence of full- or off-scale readings if its range is exceeded.
- Have a means of field checking its operability.
- Have a means of indicating when it is inoperable.
- Be small, light, and rugged, particularly if it is a portable instrument.
- Be relatively trouble-free and require only infrequent, simple repair and maintenance.

(2) The characteristics of some of the more commonly used detectors are summarized below.

(a) Ionization chambers. Ionization chambers measure dose and dose rate from gamma and X-rays. A typical ionization chamber that measures total dose is the pocket dosimeter, which is the size of a large fountain pen. It has a chamber containing two electrodes, one of which is a quartz fiber loop free to move with respect to its mounting.

Radiation entering the chamber causes ionization within the sensitive volume. The distance the fiber moves is proportional to the dose received in the chamber. Instruments of this type are sensitive to severe shock and humidity and small enough to be worn comfortably. The great advantage of this type of instrument is that it can be read at any time without the aid of a supplementary charger-reader by simply holding it up to a source of light and looking into it.

(b) Geiger-Mueller counter. Geiger-Mueller counters are normally used for detecting single ionizing events which take place within the sensitive volume of the counter. They are very rugged and sensitive to low levels of radiation. They are usually equipped with audible detection of radiation in the form of "clicks." Geiger-Mueller counters detect gamma photons or beta particles; however, the detection of gamma rays is less efficient than of beta particles. A discriminating shield is usually provided with Geiger-Mueller instruments which when opened admits both beta and gamma radiation. With the shield closed, only gamma is admitted. Use of the shield may permit qualitative differentiation between ionization caused by beta particles and that produced by gamma photons.

(c) Proportional counters. Proportional counters are used to detect one type of radiation in the presence of other types of radiation, or to obtain output signals greater than those obtainable with ionization chambers of equivalent size. Proportional counters may be used to either detect events or to measure absorbed energy (dose), because the output pulse is directly proportional to the energy released in the sensitive volume of the counter. Proportional counters are widely used for the detection of alpha particles, neutrons, and protons.

(d) Scintillation counters. A scintillation counter combines a photomultiplier tube with a scintillating material that may be a crystal or other phosphor (solid, liquid, or gas). Light pulses produced in the scintillator by radiation releases photoelectrons from the cathode of the photomultiplier tube, which then initiates pulses of current that can be counted. Scintillation counters are available that can detect alpha and beta particles, gamma rays, neutrons, protons, and electrons. The most common for field use are those employed as alpha counters or as gamma detectors. Although very energy dependent, scintillation counters are more efficient at detecting low level gamma backgrounds than are Geiger-Mueller counters.

(e) Chemical dosimeters. Chemical dosimeters are systems in which measurable chemical changes are produced by ionizing radiation. Radiation produces acids in the system, the amount of which can be determined from visible color changes, or, more accurately, by titration or pH readings. Most chemical systems of practical size are useful only for gamma doses of hundreds to millions of cGy. However, smaller volume detectors can be found which measure doses in the range of a few cGy to several thousand cGy.

(f) Photographic emulsions. Photographic emulsions are frequently used as detectors. The film badge has been the most common dosimeter in use, but is tending to be replaced by thermoluminescent dosimeters (TLD). The film badge uses the effect of radiation on photographic film to record doses. After film developing, the optical density is compared to a film calibration curve, and a measure of exposure dose is obtained. As the exposure dose increases, the optical density of the emulsion increases. At least two different types of film are employed to cover a wide-exposure range; a low exposure film, 0.02 cGy to 2 cGy and a high-

exposure film, 1 cGy to 1,000 cGy. Metal filters such as aluminum, copper, and cadmium-tungsten, are used to increase the accuracy in the reading. The heavy metal filter also intensifies the gamma radiation interaction. Beta radiation is evaluated by observing the density change to a portion of film that is not covered by a filter. Film badges or TLDs are widely used as they provide an accurate means of recording radiation exposure at a low cost. Their disadvantage is that heat, moisture, and aging will cause a natural change in the films optical density.

(g) Thermoluminescent dosimeters. Thermoluminescent dosimeters detect radiations by the formation of a metastable crystalline structure with the valence electrons. Ionizing radiation excites valence electrons to a state within the crystal structure which can be detected by heating the crystal. When heated, the electrons are released from these traps and return to their lowest energy state with the release of light. The amount of light released is proportional to the radiation exposure. Radiophotoluminescent (RPL) glass is a dosimeter material that will luminesce following an excitation pulse of ultraviolet light if it has been exposed to ionizing radiation. This effect is caused by radiation induced changes in the glass crystalline electronic structure. Although other materials also exhibit this property, silver activated RPL glass has found the greatest application in X and gamma radiation dosimetry. The sensitivity depends on the type and manufacturer selected, and ranges from 0.01 cGy to several million cGy. This type of dosimeter cannot be zeroed; it gives a total cumulative dose reading that fades only very slowly with time. Silicon diodes are most useful for high-energy neutron dosimetry. Neutrons reacting in the diodes cause displacement of atoms in the silicon crystal which results in a relatively permanent and measurable change in its electronic resistance. These dosimeters are almost totally insensitive to X and gamma radiation and have a practical range of 1 to 1,000 cGy depending on the type selected.

(3) Some of the common physical dosimeters used by the US Army are described in Tables A-1 and A-2 below:

Table A-1. Detectors in Dynamic Systems

Detector Type	Description	Examples	Detector in the Army
Gas Ionization	Gas filled chambers. Radiation causes ionization in the gas. Current is measured or charged pulses are counted. Various detectors are used, depending upon their operating voltage.	-Ionization chamber -Proportional counter -Geiger Muller counter	- Ludlum 2350 (43-68 gas flow) - AN/PDR 27, 77, ANVDR 2, others.
Scintillation	Literally a flash of light. Contains a scintillator or phosphor, in which radiation produces light and a photomultiplier tube (PMT) to detect the light and produce and electrical signal. Specific scintillators are used for different radiation types.	- ZnS (alpha) -Organic Scintillators (beta) -NaI (gamma).	- AN/PDR 56, 77 - AN/PDR 77, Ludlum 2350, EG&G Dart.
Semiconductors	Current flow in a solid crystal operated as a diode. Uses for X and gamma radiation and charged particles.	-Ge -Si	- Canbera

Table A-2. Passive Detectors

Detector Type	Description	Example
Ionization Chamber	Similar to the gas ionization principle, but it is initially charged and the cumulative discharge is measured rather than the current flow.	Ion chamber, IM-9E/PD, IM-93, IM-147.
Chemical	Exposed and then developed. Darkening is related to the previous radiation exposure.	Photographic film.
Chemical	Chemical change is proportional to radiation dose. A solid (glass or plastic) or liquid (solution) chemical system is exposed and then the chemical change (oxidation, reduction, radiolysis, etc.) is measured.	Chemical Dosimeter.
Thermo-luminescence	Thermally stimulated luminescence. After exposure, it is read out by heating and measuring the emitted light.	Thermo-luminescence Dosimeter (TLD).
Luminescence	Photo stimulated luminescence. After exposure, it is read out by stimulating with UV light and measuring the emitted visible light.	Photoluminescence AN/PDR-76.

A-3. Biological Dosimetry

A crude estimate of absorbed dose can be obtained from the clinical presentation and assessment of the response of actively proliferating cell systems to radiation. Uncertainties in dose estimates arise largely from the high variability between individuals and other factors such as infection. Generally, the sensitivity of these bio-indicators is poor; and, given the transient nature of the signs and symptoms in sub-lethal doses, their clinical usefulness is limited. A biological dosimetry is recommended in cases where medical treatment decisions are warranted. Knowledge of the distribution heterogeneity of the absorbed dose is particularly important with respect to medical treatment decisions for patients exhibiting radiation-induced bone marrow syndrome. Reliable biodosimetry is also necessary for validation of suspected low-level radiation exposures.

a. Analysis of Cytological Assays - Chromosome Aberrations. The analysis of chromosomal aberrations in peripheral blood lymphocytes is a biological dosimetry method that is widely used to assess radiation dose. Even in partial body exposures, chromosome damage is an excellent indicator of the absorbed dose. Many types of chromosomal aberrations may appear in lymphocytes following exposure to radiation, but dicentric (a chromosome with two centromeres) is a well-accepted biomarker for ionizing radiation exposure and is used to estimate radiation dose. The incidence of dicentrics in blood lymphocytes for the general population is 1 in 1,000 to 2,000 metaphases. Human T-lymphocytes have a long half-life; and a small proportion of them survives for decades. The frequency of dicentrics following exposure remains fairly stable up to a few weeks. After acute partial exposure, the irradiated lymphocytes rapidly mix with unirradiated blood, and equilibrium is reached within 24 hours.

(1) *Dicentric assay--technical aspects*

(a) Sample collection. Ten to 15 ml of blood from the exposed subject is collected in a heparinized blood collection tube. The blood lymphocytes are isolated in the cytogenetic laboratory and are stimulated to grow in culture. Cell proliferation is arrested in the

first metaphase following stimulation; and metaphase spreads are prepared. The stained first-division metaphase spreads are observed under a microscope for enumerating dicentrics.

(b) Dose-response calibration curves and factors influencing dose estimation. *In vitro* and *in vivo* irradiation of blood lymphocytes produces similar yields of dicentrics per Gy. Therefore, observed levels of dicentrics in an exposed individual can be related to dose by use of an in vitro established dose-response calibration curve. *In vitro* calibration curves have been generated for a variety of radiation qualities, including all those likely to be encountered in an accident scenario (see Figure A-1). Within any given laboratory, the calibration curves have proved very reproducible for lymphocytes examined in their first-division metaphase.

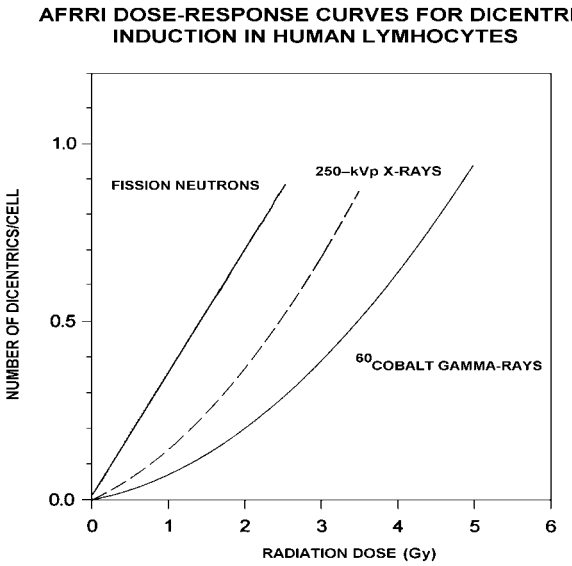

Figure A-1, Armed Forces Radiobiology Research Institute dose-response curves for dicentric induction in human lymhocytes.

(c) *Metaphases*. Two hundred to 500 metaphases are sufficient to estimate a dose at irradiation levels of medical significance. The statistical uncertainty of the dicentric yield is the main component of the 95 percent confidence limit on the dose estimate with a few hundred cells scored from an irradiated subject. The influence of the number of metaphases analyzed and the number of dicentrics scored on the 95 percent confidence limit on the dose estimate is presented in Table A-3.

Table A-3. Relation Between the Number of Dicentrics and Dose-estimates

Number of dicentrics	Number of cells analyzed	Lower confidence limit (mGy)	Upper confidence limit (mGy)	Mean dose** (mGy)
0	200	0	470	-
1	200	30	610	200
2	200	50	710	320
3	200	130	800	410
4	200	200	870	500
5	200	270	940	570
0	500	0	260	-
1	500	< 20	340	100
2	500	20	400	170
3	500	60	460	220
4	500	100	500	270
5	500	140	540	320

*Adopted from IAEA 1986.
**Whole-body gamma-ray dose

(2) *Automated analysis.* Some of the shortcomings associated with metaphase-dicentric chromosome-aberration analysis are being overcome by automation. Automated metaphase finding on slides, using image analysis techniques, significantly enhances the speed of this assay. Accurate identification of dicentrics on metaphase spreads is possible by centromere painting, either by immunoenzymatic methodology or fluorescence in situ hybridization (FISH). This helps in automated enumeration of dicentrics for rapid and reliable dose estimates; and, with increases in the number of cells analyzed, confidence limits on dose estimates are improved.

(3) *Translocation assay*. In cases of retrospective dosimetry, another subclass of chromosome aberrations, reciprocal translocations have been used for dose assessment instead of scoring dicentric chromosome aberrations. Reciprocal translocations are persistent and are formed in similar yields as dicentrics. They are typically detected using specific whole chromosome DNA hybridization probes and FISH methodology.

(4) *Premature-chromosome condensation assay*. In contrast to the enumeration of dicentrics in stimulated lymphocytes, no stimulation of cell proliferation is required for the premature-chromosome condensation assay. Polyethylene-mediated fusion of blood lymphocytes with mitotic cells results in a premature condensation of lymphocyte-chromatin material into discrete chromatid-like structures. Blood lymphocytes from normal subjects display a distribution around a mean of 46 prematurely condensed chromosomes (PCCs). Increases in the number of PCCs are evident after exposure to radiation and 24 hours of repair. Based on the increase in the number of PCCs, dose estimates can be obtained within 48 hours of receipt of blood in the laboratory. Radiation induced mitotic delay does not interfere with the assay since it is performed on interphase nuclei and does not require cell division. The method is envisioned to be applicable after partial body and/or supra-lethal exposure.

(5) *Micronucleus assay*. A micronucleus is a small chromatin body seen outside the nucleus of a daughter cell following cell division. A micronucleus is formed as a result of unincorporated chromatin material that largely arises from acentric fragments. The frequency of micronuclei in an exposed individual's blood lymphocytes is dose dependent, and therefore, can be used as a biodosimeter. However, micronuclei are not specific to radiation exposure. Discrimination between total and partial-body exposure is more difficult. High doses of radiation interfere with cell division. High base-line frequency and age dependency make the reliability of this assay questionable.

(6) *Biochemical endpoints-- deoxyribonucleic acid* s*ingle strand breaks and base alterations.* Deoxyribonucleic acid single-strand breaks (SSB) and base alterations are formed following exposure to ionizing radiation. The dose-response relationship for SSB and altered-base yields and the effect of repair at various times post-exposure are well known for various nucleated blood-cell components and can be used for dose assessment.

b. Biophysical Assays--Electron-Spin Resonance. Persistent free radicals formed in solid matrix biomaterials (dental enamel, nail clippings, and hair) from exposed personnel can be detected via electron-spin resonance. These measurements can provide reliable biophysical dose estimates as well as partial-body exposure information. In some circumstances, certain materials from clothing, particularly hard plastics and buttons may be measured and an estimate of absorbed dose made.

GLOSSARY

Section I. ABBREVIATIONS AND ACRONYMS

A_0	original activity
ABMT	autologous bone marrow transplantation
AEC	Atomic Energy Commission
AFAP	artillery-fired atomic projectile
AFRRI	Armed Forces Radiobiology Research Institute
Allo-BMT	allogeneic-bone marrow transplant
AMALS	
AMEDDC&S	Army Medical Department Center and School
amu	atomic mass unit
ANC	absolute neutrophil count
APC	armored personnel carrier
ARS	acute radiation syndrome
ATC	air transportable clinic
ATH	air transportable hospital
atm	atmospheres of pressure
BAS	battalion aid station
BDO	battle dress overgarment
BDU	battle dress uniform
BEIR V	Biological Effects of Ionizing Radiations, 5th Consensus Summary
Bq	becquerel
BW	biological warfare
BWR	boiling water reactor
C	Celsius/carbon
C2	command and control
C4I	command, control, communications, computers, intelligence
CaEDTA	calcium ethylenediaminetetraacetate
CAM	chemical agent monitor
CBF	cerebral blood flow
cc	cubic centimeter(s)
CFU	colony-forming units
cGy	centigray (0.01 Gy = 1 rad)
ChRS	chronic radiation syndrome
Ci	curie
CINC	Commander in Chief
CIWS	close-in weapon system
cm	centimeter(s)
cmv	cytomegalovirus
CNS	central nervous system (syndrome)
Co	cobalt

CONOPS	contingency operations
CONUS	continental United States
CPS	collective protection shelter
CRTS	casualty receiving and treatment ship
CRS	cutaneous radiation syndrome
CSF	cerebrospinal fluid
CSG	consumable supply group
CSH	combat support hospital
cSv	centiSievert (0.01 Sv = 1 rem)
CT	computed tomography
CV	cardiovascular
CV/CVN	aircraft carrier designation
CW	chemical warfare
DA	Department of the Army
DECON	decontamination
DMSA	meso-2,3,-dimercaptosurrinic acid
DMPS	2,3 dimercapto-1-propanesulfonic acid
DNA	deoxyribonucleic acid
DNBI	disease and nonbattle injury
DOD	Department of Defense
DRF	dose reduction factor
DTPA	diethylenetriaminepentaacetic acid (pentetic acid)
DU	depleted uranium
E	energy
Ebq	exabecquerel (10^{18}Bq)
EDTA	ethylenediaminetetraacetic acid (edetic acid)
EMEDS	
EMG	electromyography
EMP	electromagnetic pulse
EMT	emergency medical treatment
Epo	erythropoietin
ER	enhanced radiation
erg	electroretinogram
ETI	early transient incapacitation
ETI-PD	early transient incapacitation and performance decrement
Ev	electron volt
F	Fahrenheit
FAST	forward area surgical team
FBR	fast breeder reactor
FDA	Food and Drug Administration
FF	fission fragment(s)
FFP	fresh frozen plasma
FM	field manual

FST	forward surgical team
GCR	gas-cooled reactor
G-CSF	granulocyte-colony stimulating factor
GI	gastrointestinal
GLCM	ground-launched cruise missile
gm	gram(s)
gm/dl	gram/deciliter
GM-CSF	granulocyte macrophage-colony stimulating factor
GTX	granulocyte transfusions
GU	genitourinary
GVHD	graft-versus-host disease
Gy	gray (1 Gy = 100 rads)
H	hydrogen
HA	heavy armor
He	helium
HE	high explosive
HEPA	high efficiency particulate air
HEU	highly enriched uranium
HLA	human leukocyte antigen
HOB	height of burst
HSS	health service support
HVL	half-value layer
HWR	heavy water reactor
I	iodine
IAEA	International Atomic Energy Agency
IATA	International Air Transportation Association
IAW	in accordance with
ICRP	International Council on Radiation Protection
ICU	intensive care unit
ICW	intermediate care ward
IFN	interferon
IG	immunoglobulin
IL	Interleukin
IM	intramuscular(ly)
IND	investigational new drug
IV	intravenous(ly)
JSLIST	Joint service lightweight integrated suit technology
K	potassium
kBq	thousand becquerels
keV	thousand electron volts
kg	kilogram(s)

KGB	Komitet Gosudarstvennoy Bzopasnosti
km	kilometer(s)
kPa	kilopascals
KT	kiloton(s)
kVp	kilovolts (peak)
LAW	light antitank weapon
LD	lethal dose
LET	linear energy transfer
LIHOPO	a hydroxypridizone ligand
LWGR	light water graphite reactor
m	mass
MASF	mobile aeromedical staging facility
MCRP	Marine Corps Reference Publication
MCW	minimal care ward
MES	medical equipment sets
MeV	million electron volts
mg	milligram(s)
MGDF/Tpo	megakaryocyte growth and development factor/thrombopoietin
mGy	milligray (0.001 Gy; 10 mGy = 1 rad)
ml	milliliter(s)
mm	millimeter(s)
mm3	cubic millimeter
MOLLE	modular lightweight load-carrying equipment
MOPP	mission-oriented protective posture
MOX	mixed oxide
mph	miles per hour
mrem	milliroentgen equivalent in man/mammal
MRI	magnetic resonance imaging
mSv	milliSievert (0.001 Sv; 10 mSv = 1 rem)
MT	megaton
MTF	medical treatment facility
MUPS	medically unexplained physical symptoms
mW	megawatt
Na	sodium
NATO	North Atlantic Treaty Organization
NAVMED P	US Navy Medical Publication
NBC	nuclear, biological, and chemical
NCRP	National Council on Radiation Protection and Measurements
NDI	nondestructive inspection
NPO	nulla per os (nothing by mouth)
NRC	Nuclear Regulatory Commission
NVD	night vision device
NW	nuclear warfare

OEG	operational exposure guidance
OR	operating room
OSHA	Occupational Safety Health Administration
PAF	platelet activating factor
PAL	permissive action links
PBSC	peripheral blood stem cell
PBSCT	peripheral blood stem cell transplantation
PCC	prematurely condensed chromosome
PCR	polymerase chain reaction
PD	performance decrement
PET	positron emission tomography
pg	picograms
PHS	Public Health Service
PIES	proximity, immediacy, expectancy, simplicity (mnemonic for treatment of psychiatric casualties)
PMN	polymorphonuclear neutrophil
PO	per os (by mouth/orally)
PO2	plutonium oxide
ppm	parts per million
PPW	patient protective wrap
PRBC	peripheral red blood cell
psi	pounds per square inch
PTSD	post-traumatic stress disorder
PTX	pentoxifylline
Pu	plutonium
PVNTMED	preventive medicine
PWR	pressurized water reactor
R	roentgen
R&D	research and development
Ra	radium
rad	radiation absorbed dose (term no longer used)
RBC	red blood cell
RBE	relative biological effectiveness
RCS	reactor coolant system
RDD	radiological dispersal device
REAC/TS	Radiation Emergency Assistance Center/Training Site
rem	roentgen equivalent in man/mammal (term no longer used)
RES	radiation exposure status
Rn	radon
RNA	ribonucleic acid
RPL	radiophotoluminescent
RTD	return to duty

SAT	serum-agglutinating titers
SD	skin dose
sec	second(s)
SI	systems international
SNM	**s**ensitive nuclear material
SPECT	single photon emission computed tomography
SSB	single-strand break(s)
SST	
STANAG	Standardization Agreement
Sv	Sievert (SI unit of roentgen dose equivalent)
T-AH	Navy hospital ship designation
TB	treatment brief
TCDO	tetrachlorodekaoxide
TDM	trehalose dimycolate
TG	technical guide
ThO	thorium oxide
TLD	thermo-luminescent dosimeter
TMP	trimethoprim
TO	theater of operations
TNF	tumor necrosis factor
TNT	trinitrotoluene
TPN	total parenteral nutrition
TSH	thyroid-stimulating hormone
TSST-1	toxic shock syndrome toxin-1
µ	microns
µCi	microcurie
U	uranium
UO2	uranium oxide
US	United States
USAF	United States Air Force
USAMRIID	United States Army Medical Research Institute for Infectious Diseases
USTRANSCOM	United States Transportation Command
UTC	unit type code (United States Air Force)
VA	Department of Veterans Affairs
vWf	von Willenbrand factor, clotting factor 8
VS	vital signs (?)
WHO	World Health Organizations
WMD	weapons of mass destruction
WMDT	wartime medical decontamination team
W_R	radiation weighting factor

Z/A	ratio of atomic number to atomic mass
ZnDTPA	zinc diethylenetriaminepentaacetic acid

Section II. DEFINITIONS

acute radiation syndrome	a combination of clinical syndromes occurring in stages, during a period of hours to weeks after exposure, as radiation injury to various tissues and organs is expressed.
afterwind	wind currents that are created near a nuclear explosion by the updraft accompanying the rise of the fireball and that travel toward the center of the blast.
agranulocytosis	a complex of symptoms characterized by marked decrease in the number of granulocytes and by lesions of the throat and other mucous membranes, of the gastrointestinal tract, and of the skin.
airburst	the explosion of a nuclear weapon at such a height that the expanding fireball does not contact the earth's surface.
alpha particle	a positively charged particle ejected from the nucleus of a radioactive atom, being a high-speed ionized atom of helium. A stream of these particles constitutes alpha rays.
aplasia	lack of development of an organ or tissue, or of the cellular products from an organ or tissue.
atomic number	the number of protons in an atomic nucleus.
beta particle	an electron emitted from an atomic nucleus during beta decay.
bioassay sampling	indirect means of measuring contamination in body tissue or fluids from which body burden is extrapolated.
blast wave	a pulse of air in which the pressure increases sharply at the front propagated by the explosion.
bremsstrahlung	the process by which a beta particle emits an X-ray photon during its interaction with an atomic nucleus.
carcinogenesis	the development of cancer; various malignant growths that manifest invasiveness and a tendency to metastasize to another location.
cataractogenesis	the development of cataracts; opacity of the lens causing blindness.

cerebral anoxia	absence of an oxygen supply to the brain despite adequate perfusion of the tissue by blood.
congestion	excessive or abnormal accumulation of blood in a tissue or organ.
critical mass	the minimum amount of a fissionable material needed to sustain a nuclear chain reaction for a given set of circumstances.
critical organ	body organ with an affinity for a particular substance and in which that substance concentrates.
cytokine	a nonantibody protein released by one cell population that acts as an intercellular mediator on another cell population(s). Synthetic cytokines are metabolically active in pico-molar concentrations.
decontamination	reduction or removal of radioactive contamination from a structure, area, object, or person.
delayed fallout	radioactive fallout that returns to earth later than 24 hours after a nuclear detonation; usually ascends into the stratosphere and is distributed worldwide.
desquamation	the shedding of epithelial elements, chiefly of the skin, in scales or small sheets; exfoliation.
dose rate	a measure of the amount of ionizing radiation an individual is exposed to, per unit of time; commonly expressed in units of gray (or rads) per hour.
deterministic effect	one that occurs after a certain dose threshold, with the severity of the effect determined by the dose; example: cataractogenesis.
dynamic pressure	air pressure that results from the wind behind the shock front of a blast wave.
early fallout	radioactive fallout that returns to earth within 24 hours after a nuclear detonation; also referred to as local fallout.
edema	the presence of abnormally large amounts of fluid in the intercellular tissue.
exposure	a measure of the number of ionizations produced by gamma or X-rays in a volume of air; expressed in units of roentgen.

fallout	the process or phenomenon of the descent to the earth's surface of particles contaminated with radioactive material from the radioactive cloud produced by a nuclear detonation.
fireball	hot gases that form a luminous sphere after a nuclear explosion.
fission	the process of splitting the nucleus of an atom into parts.
fission products	a general term for the complex mixture of isotopes produced as a result of nuclear fission.
flash burn	a burn caused by excessive exposure of the skin to thermal radiation.
free-in-air-dose	radiation that would be measured in air at a certain point. Military tactical dosimeters measure free-in-air-doses.
gamma ray	electromagnetic radiation of short wavelengths emitted by the nucleus of an atom during a nuclear reaction. They consist of high-energy photons, have no mass and no electric charge, travel with the speed of light, and are usually associated with beta rays.
granulocyte	any cell containing granules in its cytoplasm, especially a leukocyte (neutrophil, basophil, or eosinophil).
granulocytopenia	agranulocytosis; a symptom complex consisting of a marked decrease in the number of circulating white blood cells, with lesions of the throat and mucous membranes.
hematopoietic	pertaining to, or effecting, the formation of blood cells.
hemorrhage	the escape of blood from the vessels; hemorrhages, classified according to size: petechiae (pinpoint) or ecchymoses (larger than 1 centimeter) in diamerter).
hyperpyrexia	a highly elevated body temperature.
hypotension	abnormally low blood pressure.
induced radiation	radioactivity produced in certain materials as a result of the capture of neutrons, which is accompanied by the formation of unstable nuclei.
ingestion pathway	route for internalization of radioactive contaminants; the pathway most accessible for decontamination.

inhalation pathway	primary pathway for internalization of radioactive contaminants.
ionization	the process of stripping electrons from their atomic orbits by radiation.
isotope	one of two or more atoms whose nuclei have equal numbers of protons but different numbers of neutrons.
late effect	a biological effect that occurs long after radiation exposure ends; example: cancer.
lymphocyte	a mononuclear leukocyte; chiefly a product of lymphoid tissue and participates in humoral and cell-mediated immunity.
Mach stem	the shock front formed by the merging of the primary and reflected shock fronts from an explosion.
malformation	a birth defect; an abnormal structure or form; example: small head.
morbidity	the ratio of sick to well individuals in a community; the sick rate.
mortality	the ratio of people who die to those who survive irradiation; the death rate.
nadir	the point at which a blood count drops to, or closest to zero, before beginning to increase.
neutron	an electrically neutral or uncharged particle of matter existing along with protons in the atoms of all elements except the mass 1 isotope of hydrogen.
neutropenia	a decrease in the number of neutrophilic leukocytes in the blood.
nosocomial	pertaining to or originating in a hospital.
nuclear/radiological agent	traditionally, uranium or plutonium used to produce a nuclear detonation via the fission or fusion process. The fuel is compressed into a given volume to cause supercriticality. The major products include blast effects, heat, nuclear radiation, and fallout.
nucleated blood cell	a blood cell that contains a nucleus, to include white cells and reticulocytes.
nucleon	a proton or neutron as part of an atomic nucleus.

nuclide	an atomic nucleus with a specific energy state and specific number of protons and neutrons.
orbital excitation	change in energy level of an orbital electron that occurs when the energy lost by the incident radiation is insufficient to cause ionization.
overpressure	the transient pressure that is created by the shock wave of an explosion and exceeds the ambient pressure; expressed in pounds per square inch.
pathognomonic	specifically distinctive or characteristic of a disease or pathologic condition; a sign or symptom on which a diagnosis can be based.
perceived threat	a threat that is experienced by a person subjectively and out of proportion to the real threat or physical danger.
phagocytosis	the engulfing of microorganisms, other cells, and foreign particles by phagocytes.
pressor	tending to increase blood pressure.
prodrome	a premonitory symptom or precursor; a symptom indicating the onset of a disease.
Prussian blue	ferric ferrocyanide; a chemical that is not absorbed by the gastrointestinal system and is an effective means to reduce the body's uptake of cesium, thallium, and rubidium; approved as an investigational new drug by the United States Food and Drug Administration with the license held by Radiation Emergency Assistance Center/Training Site.
radioactive cloud	an all-inclusive term for the cloud of hot gases, smoke, dust, dirt, and debris from a weapon and the environment. The cloud is carried aloft in conjunction with the rising fireball produced by the detonation of a nuclear weapon.
radioactive contamination	radioactive material in an undesirable location such as on structures, areas, objects, or personnel.
radionuclide	a radioactive nuclide; one that disintegrates with the emission of particulate or electromagnetic radiations.
rainout	the removal of radioactive particles from a nuclear cloud by precipitation when the cloud is within a rain cloud.

reproductive death	the loss of the ability to reproduce. Many organs must have cells that can reproduce to function. Thus, even though injured cells may remain biologically viable, reproductive death may cause irreversible organ damage.
scavenging	the selective removal of material from the radioactive cloud by inert substances, such as precipitation, introduced into the fireball.
sepsis	the presence of pathogenic microorganisms (bacteria) or their toxins in the blood or other tissues.
septicemia	systemic disease associated with the presence and persistence of pathogenic microorganisms or their toxins in the blood.
skin permeability	the rate at which the skin absorbs a liquid; expressed as a coefficient. The lower a substance's coefficient, the greater the rate of absorption.
Specific ionization	number of ion pairs per unit distance formed along the path of a particle, often expressed as ion pairs per centimeter.
stochastic effect	an effect that has no-dose threshold and for which the severity of the effect is not dose-related, although its probability is; example: carcinogenesis
stratosphere	a relatively stable layer of the atmosphere extending from the tropopause to an altitude of about 30 miles.
subsurface burst	the explosion of a nuclear weapon beneath the surface of the earth.
syndrome	a set of symptoms that occur together.
synergistic	acting together to enhance the effect of another force or agent.
thermal radiation	electromagnetic radiation (infrared, visible, and ultraviolet) emitted from the fireball of a nuclear explosion as a consequence of high temperatures.
total parenteral nutrition	by injection through some route other than the alimentary canal.
tropopause	the layer of the earth's atmosphere that separates the troposphere from the stratosphere.
troposphere	the layer of the earth's atmosphere extending from the surface up to the tropopause, normally about 25,000 feet in temperate zones and about 55,000 feet in the topics.

washout	the removal of radioactive particles from a nuclear cloud by precipitation when the nuclear cloud is below a rain or snow cloud.
weapon debris	highly radioactive material consisting of fission products, various products of neutron capture, unspent fuel, and shards of bomb casing that remain after a nuclear explosion.
wound contamination	the presence of a radioactive substance in a wound, whether an abrasion, puncture, or laceration; condition in which the loss of intact skin increases the risk that the contaminant will be absorbed.

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